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CONTENTS

PAGES

Treatment (*concluded*) 97–101
Specific (Tuberculin, etc.) (concluded) 97– 97
Drugs and Chemotherapy 97–100
Dietetic 100–101
Diseases Other than Tuberculosis 101–123

Ferrán's Treatment of Tuberculosis.—Ferrán isolated from pure tubercle-bacillus cultures non-acid-fast streptobacilli which seem to be closely related to colon bacilli. These "alpha-bacilli" are not tuberculogenic, but when they are transmitted in continuous animal passage they recover all the characteristics of the classical tubercle bacillus. Ferrán believes that the disease tuberculosis is caused by a mutation of the alpha-bacillus into the acid-fast form, and that the former occurs as a harmless saprophyte in the intestinal canal. He believes that this mutation can be prevented by immunizing the body against the alpha-bacillus, and that the same treatment is effective therapeutically in clinical disease. The author treated 108 patients with the alpha-bacillus vaccine, and with anti-alpha serum. He observed striking improvements, such as disappearance of bacilli from sputum, return of temperature to normal, improvement of the haematological findings and of the general condition. Improvement is reported in 96 per cent of patients classed as Turban I, in 70 per cent Turban II, in 46 per cent Turban III, and in 65 per cent of extrapulmonary lesions. The treatment is contraindicated in cases of generalized tuberculosis, in the presence of energy and of massive haemoptysis.—*Tuberkulosetherapie nach Ferrán, O. Felsenfeld, Wien. klin. Wchnschr., August 21, 1930, xliii, 1066.*—(M. *P.*)

Drugs in Treatment of Tuberculosis.—Drugs have recognized value in certain symptoms and complications of pulmonary tuberculosis, but no known specific effect on the disease. Copper salts have fallen into disuse because of severe reactions. In 1890 Koch found gold preparations effective *in vitro*, but they were too toxic for use in human beings. Sanocrysin inhibits growth of tubercle bacilli in culture, but this effect is lost upon the addition of serum. Clinically, it produces no violent reactions in the proper dosage and the sputum and bacilli decrease in amount. Heaf found initial improvement in clinical condition and sedimentation rate, but 75 per cent of his cases later relapsed. The author's method of dosage is given in detail, but his results are not given. References to studies of other gold preparations are given. Good results have been described following the administration of calcium. Half the calcium chloride injected intravenously is excreted within three hours, and the remainder within three days. In one case Elving found that the coagulation rate of the blood increased from five and a half minutes to half a minute after the intravenous injection of 20 cc. of a 15 per cent solution of calcium chloride. Calcium should be given over long periods of time, and preferably by mouth and intramuscularly. Codliver oil and parathyroid should be given with it. In whatever form iodine is given, it forms an iodide in the blood. It is of most benefit if bronchitis is present. Sodium cacodylate probably has no effect except as a tonic. Creosote is harmful in large doses and useless in small ones. Often

there seems to be a chronic deficiency of vitamines in tuberculosis. There is a greater need for vitamines at puberty than at any other age.—*Critical Survey. Drug Treatment in Pulmonary Tuberculosis, L. S. T. Burrell, Brit. J. Tuberc., July, 1930, xxiv, 134.*—(*R. M. F.*)

Treatment of Pulmonary Tuberculosis with Gold.—The following gold preparations, in the order of their decreasing toxicity, were used in 150 cases of pulmonary tuberculosis: triphal, sanocrysin, solganol and lopion. Solganol has the slightest action upon the kidneys but was toxic for the intestines. Lopion was least toxic and seemed to be preferred for this reason. Therapeutic results could only be obtained with large doses. Most suited for gold therapy are the early processes and cases of indurated phthisis, while highly active exudative cases are unsuitable. Of importance to the theory of the gold action is the fact that haematogenous processes are uninfluenced and this would seem to indicate that the gold therapy is not a genuine chemotherapy, but rather its action is that, in the sense of Feldt, upon the reticuloendothelial system with the production of antibodies and a detoxication.—*Über Behandlung der Lungentuberkulose mit den neuen Gold-präparaten, A. Fischer, Zeitschr. f. Tuberk., 1929, lii, 465.*—(*H. J. C.*)

Sanocrysin.—Acute bilateral pulmonary tuberculosis with unilateral pneumothorax, subacute cases that are unresponsive to treatment and are slowly progressive, chronic fibroid cases with exacerbations, and any case with many tubercle bacilli in the sputum may all be treated with sanocrysin. The dosage at the beginning should be 0.1 gm. and should gradually be increased to 1.0 gm., and this given weekly until a total of 5.0 or 6.0 gm. has been administered. A persistent albuminuria will follow larger doses. Immediate results are encouraging, although relapses occur frequently. Many patients lose the bacilli from the sputum and this is the most striking result of the treatment. Bad results may be avoided by use of small doses. These are a rash, even to an exfoliative dermatitis, diarrhoea and transient albuminuria. The initial improvement has seemed in some cases to lead to permanent improvement.—*Sanocrysin in the Treatment of Pulmonary Tuberculosis, L. S. T. Burrell, J. State Med., May, 1930, xxxvii, 281.*—(*H. S. W.*)

Sanocrysin.—At Vejlefjord Sanatorium, a group of 26 patients with clinical tuberculosis were treated with sanocrysin. These patients, all with acute phases of fresh, spreading disease, responded well to sanocrysin, and showed such an immediate effect and uniformity of results as to clinically correspond to Möllgaard's experimental results. Sanocrysin, suitably administered, exerts a specific effect by cutting short recent pulmonary disease. In the large majority of patients, in whom early and late phases of the phthisis are intermingled, by combining sanocrysin and collapse treatment, there have been results which could not have been obtained by either method alone, sanocrysin acting on the fresh disease, the older fibrocavernous changes being healed by collapse. On pronounced "tertiary" phases, sanocrysin alone has no remarkable effect.—*The Value of Sanocrysin in Relation to the Different Phases of Pulmonary Tuberculosis, J. Gravesen, Tubercle, February, 1930, xi, 193.*—(*W. I. W.*)

Sanocrysin in Experimental Tuberculosis.—An affinity of the gold preparation for tubercle bacilli *in vivo* was not determined, but could be noted with tuberculous tissue, in that tuberculous lung contained more gold in sanocrysin-treated rabbits than the lungs of controls or in those in which the sanocrysin treatment inhibited the tuberculous process. The effect of sanocrysin was also more striking the earlier the treatment was instituted after infection. At later intervals or when low virulent strains of tubercle bacilli were used there was no effect.—*Die Sanocrysinbehandlung bei experimenteller Tuberkulose. II. Kaninchentuberkulose, T. Madsen and J. R. Mörch, Zeitschr. f. Hyg., 1929, cix, 224.*—(*H. J. C.*)

Sanocrysin in Experimental Tuberculosis.—In order to explain the negative findings of Bang as compared to those of Madsen, a coöperative experiment was performed on 166 rabbits, using a bovine-bacillus culture *A* and another culture *B*, which had been perpetuated since 1924 in two different laboratories. Culture *B* had suffered a loss in virulence during this interval. The experiment, which was interrupted by an intercurrent infection, resulted negatively with sanocrysin, and Madsen and Mörch attribute this to a changed behavior of the tubercle-bacillus culture used for infection.—*Die Sanocrysinbehandlung bei experimenteller Tuberkulose. III. Kaninchentuberkulose, O. Bang, T. Madsen and J. R. Mörch, Zeitschr. f. Hyg., 1929, cix, 243.*—(*H. J. C.*)

Blood Changes Following Sanocrysin.—A preliminary report is given on the blood

changes in 4 patients with chronic pulmonary tuberculosis and in 2 normal persons, following injections of sanocrysin. In all persons there was frequently a steady fall of red and white cells for two to four days after each injection. An increase in monocytes occurred after each injection. In the nontuberculous subjects and in the tuberculosis patients which were helped by the treatment, there was a lymphocytosis from two to five days after injections. In those patients in which the sanocrysin produced a bad effect on the disease, the lymphocytes decreased from the beginning of the treatment. Eosinophilia was frequently observed. These changes resemble those occurring during the spontaneous cure of the disease and suggest that the chief action of sanocrysin in pulmonary tuberculosis is to stimulate the curative processes already going on in the body.—*Blood Changes Following Injection of Sanocrysin in Pulmonary Tuberculosis, T. A. Hughes and D. L. Shrivastava, Brit. M. J., August 16, 1930, no. 3632, 248.*—(*M. E. P.*)

Treatment of Pulmonary Tuberculosis with Calcium.—Calcium chlorate and calcium gluconate (Sandoz) was used subcutaneously and intramuscularly in 78 cases. Of those treated, 59 improved, 16 were uninfluenced and 3 did not tolerate the calcium. Among 61 open cases of old tuberculosis 7 became closed. Subfebrile temperatures are beneficially influenced while febrile cases are not. The author maintains that the calcium neutralizes the chemical acid combinations of the blood during fever. Calcium had no inhibitory effect upon exudates, although some benefit was noted.—*Die intravenöse Kalkbehandlung der Lungentuberkulose, F. Becker, Zeitschr. f. Tuberk., 1929, liii, 198.*—(*H. J. C.*)

Effect of Calcium on Fatiguability in Tuberculosis.—It has been found that the fatiguability of the muscles of accommodation is a rather reliable index of the general fatiguability. Reading small print at the shortest possible distance requires a maximal contraction of the ciliary muscle. The length of time that this maximal contraction can be maintained, that is, that vision remains clear at the minimal distance, represents a measure for the fatiguability. With this test it was determined in a number of patients what effect "kalzan" (a sodium-calcium-lactic acid compound) has on the fatiguability of tuberculous patients. It was found that after a few days' medication, the fatiguability decreased markedly. The effect of calcium lactate and calcium chloride was qualitatively the same, but quantitatively much less marked.—*Ueber die Wirkung vermehrter Kalkzufuhr auf die Adynamie der Lungentuberkulösen. Versuch einer neuen Methode der Ermüdbarkeitsmessung, H. Stein, Wien. klin. Wchnschr., April 17, 1930, xliii, 496.*—(*M. P.*)

Calcium Therapy and Haemoptysis.—During the course of intensive intravenous calcium therapy, 7 patients had profuse pulmonary haemorrhages. In one of the cases haemoptysis had never occurred prior to the treatment. In another patient, physical signs of cavity manifested themselves shortly after the haemorrhage. It is quite possible that by increasing the coagulability of the blood by means of calcium salts, thrombosis occurs more readily and thrombotic infarction ensues which in turn gives rise to haemorrhage and pneumonic reactions. Another theory holds that calcium predisposes to haemorrhage by relaxing bronchospasm and giving rise to a hyperaemia of the bronchial mucous membrane.—*L'uso del calcio puo' talora essere di danno nelle emottisi?, Arturo Campani, Tubercolosi, xxi, April, 1929, 135.*—(*U. E. Z.*)

Calcium and Iodine in Treatment of Pulmonary Tuberculosis.—The use of calcium and iodine in the treatment of four patients with pulmonary tuberculosis is described. Three patients received weekly intramuscular injections of 0.5 cc. of calcium (named collosal calcium by the author) and 3 minims of tincture of iodine (without potassium iodide) in a dessertspoonful of cod-liver-oil cream orally thrice daily. The fourth patient received iodine in codliver-oil cream, as above, and calcium lactate (5 grains daily) by mouth. In all patients the number of bacilli in the sputum was decreased, weight increased, fall in temperature noted, and marked improvement in physical findings and general health found. —*Treatment of Pulmonary Tuberculosis on the Lines of Mineral Deficiency, K. Fraser, Brit. M. J., May 24, 1930, no. 3620, 946.*—(*M. E. P.*)

Metallic Salt Therapy.—Cadmium and manganese possess a specific healing value in rabbits infected with tubercle bacilli, while only a slight action was noted with cerium, barium, aluminum, lanthinum, molybdium, platinum, nickel and samarium, the remaining 32 of 42 metallic salts tested being without action.—*Metallsalztherapie. Abschliessende Versuche über Kaninchentuberkulose, L. E. Walbum, Zeitschr. f. Tuberk., 1929, liii, 292.*—(*H. J. C.*)

Carbol-Fuchsin in Chemotherapy of Tuberculosis.—In an attempt to find a

method of staining tubercle bacilli in the lungs by way of the blood-stream the author gave an injection of 10 cc of carbol-fuchsin intravenously, and found that the normal lung tissue of rabbits was stained in animals that died one hour after injection while tuberculous necrotic tissue was not. In frozen sections tubercle bacilli were also unstained. On the other hand tuberculous guinea pigs, given 5 injections of 5 cc. of concentrated watery carbol-fuchsin in the course of 2½ weeks, revealed a number of small tubercles stained completely. As a result of these experiments the author concludes that there may be the possibility of a bactericidal chemotherapy in early cases.—*Zur Frage der Möglichkeit einer bactericiden Chemotherapie der Tuberkulose, F. Jessen, Beitr. z. Klin. d. Tuberk., 1929, lxxii, 184.*—(*H. J. C.*)

Dietary Treatment in Tuberculosis.—The so called Sauerbruch diet in tuberculosis is based on a multiplicity of investigations and observations. Herrmannsdorfer observed better healing of wounds during an acid diet; Gerson, in association with the former author, paid particular attention to the mineral metabolism; codliver oil with phosphorus was added later on, as were fresh vegetables and raw fruit in order to increase the vitamine content. The theoretical foundations of this diet are still much under discussion, especially since this diet, whose proclaimed aim is relative acidosis and alteration of the mineral metabolism, does apparently not change the pH of the blood, nor the alkali reserve, nor the ratio between sodium and calcium. Whether the empirical mixture of many inorganic salts, sold under the name of *mineralogen*, plays any important rôle in the treatment is still uncertain. Jesionek and Bommer, who reported good results with the diet in skin tuberculosis, had equally good results without the administration of mineralogen. The same uncertainty prevails in regard to the practical merits of this dietary regimen. Almost all authors agree that tuberculosis of the skin, and lupus in particular, are most effectively influenced. The number of such cases reported from various clinics is sufficient to prove the merit of the treatment, although a few recurrences have been observed. In surgical tuberculosis the reported views are conflicting; and in pulmonary tuberculosis the majority of observers reported no beneficial results. Since February, 1929, about 50 patients received the diet, strictly following Herrmannsdorfer's direction. Most of these patients had tuberculosis of bones and joints, frequently with sinuses, some had associated pulmonary lesions, foci in the urogenital system, and lupus. A brief clinical report of each patient is published. During the special dietary regimen, no other treatment was given. Although it is too early to draw definite conclusions, it must be emphasized that in almost all patients a striking and sudden improvement in the general condition set in. Within two to three months, gain in weight of 20 to 30 pounds was observed, and sinuses dried up and closed rapidly. The observations, as far as they go, are most encouraging and should prompt further and more extensive studies. In the preparation of the food, unusual care and adaptation to individual taste is of paramount importance.—*Zur Ernährungsbehandlung der Tuberkulose, P. Clairmont and A. Dimtza, Klin Wchnschr., January 4, 1930, ix, 5.*—(*M. P.*)

Sauerbruch's Dietary Treatment of Tuberculosis.—The curative principle in the regimen, as advocated by Sauerbruch, Herrmannsdorfer, and Gerson, is the lack of sodium chloride in the diet. This contention is supported by the following arguments: After a sufficiently long period of sodium-chloride starvation, the secretion of hydrochloric acid in the stomach ceases; the lack of hydrochloric acid causes an increase of putrefactive processes in the intestines with an excessive production of phenol and paracresol. This "autophenolisation" is a marked curative factor in tuberculosis. In this connection it is significant that susceptible cattle secrete no phenol compounds in the urine and relatively resistant horses excrete large amounts. Intestinal phenol production depends on a diet rich in vegetable proteins; but, if animal protein is preferred, phenol (about 250 mgm. per day) may be given by mouth with equally good results. With a diet rich in vegetable proteins, or with phenol medication, essentially the same results can be obtained as with diet free from sodium chloride.—*Welcher Factor bedingt die Heilwirkung der Sauerbruch-Herrmannsdorfer-Gersonschen Tuberkulosediät?, O. L. E. de Raadt, Wien. klin. Wchnschr., June 12, 1930, xliii, 752.*—(*M. P.*)

Treatment of Tuberculosis with Gerson's Diet.—In the diet which was originally suggested by Gerson and modified by Herrmannsdorfer and Sauerbruch, several factors may be of importance, namely, marked reduction in sodium chloride, probable increase in acidity of the tissues, codliver oil, phosphorus, and the empirical mixture of inorganic salts, which is known under the name of *mineralogen*. It is likely that this diet has a dehydrating effect, which would explain its beneficial action on skin and bone

lesions, but, since the pulmonary tissue does not contain much sodium chloride, a marked effect on pulmonary lesions would not be expected. The practical results which have been reported are sufficiently encouraging to invite further trials.—*Ist die Gerson-Diät bei Tuberkulose zu empfehlen?, W. Falta, Wien. klin. Wchnschr., January 30, 1930, xliii, 148.*—(*M. P.*)

Splenic Opotherapy in Tuberculosis.—Among the tuberculous children admitted to Armand Delille's service at the *Hôpital Hérold* from 1926 to the end of March, 1928, 26 received splenic opotherapy. All these were bacilliferous, and in all pneumothorax was either contraindicated or impossible or, having been established, could not check the progress of the disease. Most of the children were in poor general condition, with continued loss of weight, extensive roentgenographic changes, and physical signs apparently presaging a fatal termination. Some were febrile. In 4 of the 26 splenic treatment was unable to modify their course and all these resulted fatally. In 22, however, the evolution was surprising. A large proportion were cured or ameliorated. Their case histories are cited in detail, with temperature, pulse, and weight curves, and reproductions of serial roentgenographs. Twelve injections of 5 cc. per month seemed to be the usual dosage given. *Summary:* The following facts are observed: *1: Weight:* Three cases gained over 10 kilos, 3 over 8 kilos, 4 over 5 kilos, 6 over 3 kilos, 4 over 2 kilos, and 1 over 1. One case lost weight. *2: Tubercle bacilli* disappeared in 10 cases, were still present in 12. *3: Roentgenographs* showed clearing in 11, no clearing in 11. In 3 cases clearing was complete, in 4 remarkable, in 4 very definite. *4: Erythrocytes* showed in all cases counted a marked increase that averaged a million per cu. mm. blood. *5: Haemoglobin* showed comparable increases. *6:* Fever was present in 12 cases, of which 8 became apyretic and in 4 it was lowered. *7: General Results:* There were among the 26 cases, 4 deaths, 1 aggravation, 4 improved, 7 notably improved, 10 clinically cured.—*Documents à l'appui du traitement de la tuberculose par l'opothérapie splénique, M. Bayle, Rev. d. l. Tuberc., June, 1929, x, 335.*—(*A. P.*)

Treatment of Tuberculosis with Spleen.—The authors report 12 cases of pulmonary tuberculosis given a diet of spleen. The subjective findings were improved in all cases, the temperature diminished and the weight increased, while the chest findings indicated an improvement in the pulmonary process. The bacilli in the sputum diminished or disappeared. The blood picture revealed an increase in lymphocytes, monocytes and eosinophiles, while the leucocytes diminished. It is left undecided as to whether the feeding with spleen had a direct action upon the tubercle bacilli or increased the resistance of the body. The administration of spleen can be in the form of fresh tissue, whether as an injection, syrup or powder. The powdered form is considered valuable since it is easily administered and never becomes objectionable.—*Über Milzdiät bei Tuberkulösen, P. Ruttgers and A. Kamsler, Beitr. z. Klin. d. Tuberk., 1929, lxxii, 68.*—(*H. J. C.*)

Fatal Human Anaphylactic Shock.—A case of fatal anaphylactic shock after a single injection of diphtheria antitoxin is reported. The pathological anatomy is described, consisting chiefly of acute pulmonary emphysema, dilatation of the right side of the heart, general venous stasis and visceral congestion; the left side of the heart was contracted and empty. Microscopically, the changes consisted chiefly of thickening of the arteriolar walls, especially in the lungs. These anatomical changes are presented as corroborative morphological evidence to substantiate the experimental work on the pathogenesis of fatal anaphylactic shock reviewed. This consisted chiefly in overfilling of the lungs, which remained distended, a drop in the peripheral blood-pressure with a concomitant rise in the pulmonary arterial pressure, a progressive weakening of cardiac contraction, and a slowing and final stopping of the heart, with the right auricle and ventricle in diastole and very much dilated. Doubt is cast on the rôle of the thymus in such cases.—*Fatal Human Anaphylactic Shock, J. G. M. Bullowa and M. Jacobi, Arch. Int. Med., August, 1930, xlvi, 306.*—(*W. I. W.*)

Bronchial Asthma.—Death due to a paroxysm of bronchial asthma is very rare. A report is made of death occurring suddenly during a paroxysm in a patient who had been under treatment for 7 years. In this case the temperature was usually subnormal. Attempts to cause a rise in temperature caused a severe reaction with extreme cyanosis and unconsciousness. The patient died following injection of a previously effective dose of epinephrine. The cause of death in this case was determined to be asphyxia, due to a long-standing chronic bronchitis with thickening of the bronchial walls and narrowing of the lumen of the bronchi, a superimposed reaction characterized by contraction of the muscular layer with marked infolding of the mucous membrane, an outpouring of

mucus, and a flooding of the tissues of the bronchi with eosinophiles. There was no hypertrophy of the right heart, no arteriosclerosis of the pulmonary vessels, and no emphysema.—*Death from Bronchial Asthma, C. B. Wright, J. Am. M. Ass., April 19, 1930, xciv, 1218.*—(*G. L. L.*)

Bronchial Asthma.—There exists a type of asthmatic person in whom, even in the absence of other complications, antibody-formation against allergic antigens is apparently greatly reduced or nonexistent. In the absence of antigenic reproduction, such as occurs in bacterial infections, there is probably combined with this lowered faculty of antibody-formation a definite inability to excrete promptly the toxins elaborated by such allergic antigens following their complete withdrawal. Eosinophilia is usually absent in these cases. The usual diagnostic skin tests cause negative reactions, and, if pollens are used, are dangerous. In these cases there is gradual, and at times rapid development of polyantigenic sensitiveness of high degree. A small but appreciable percentage of these patients recover a certain amount of antibody-formation and relief and even freedom from symptoms after many weeks or months of persistent elimination of antigenic contact without the institution of other therapeutic measures. While there is no laboratory proof to support these contentions, the clinical results secured offer rather conclusive proof. There exists no known reason why a patient should not fail to develop antibodies to allergic as well as to bacterial antigens.—*Bronchial Asthma, I. S. Kahn, Arch. Int. Med., August 1930, xlvi, 218.*—(*W. I. W.*)

Asthma and Molds.—In a woman who had been a victim of asthma for nine years, cutaneous tests with representative plant pollens, food proteins, epidermal proteins and a stock house-dust extract were repeatedly negative. The first attack of asthma occurred after she had lived four years in a damp and musty brick house. An extract of *Aspergillus fumigatus* in a dilution of 1:5000, administered subcutaneously, gave rise to a marked local reaction and provoked the asthmatic state. In a series of 125 patients, this woman was the only one to react positively to mold reagents, an incidence of 0.8 per cent. Physical and radiographic examinations of the patient were negative. Regional mold surveys may assume an importance in the control of asthma equal to that of botanical surveys in hay-fever. Especially is this true of the grain and fruit producing section of the country.—*Asthma Due to a Mold—Aspergillus Fumigatus, H. S. Bernton, J. Am. M. Ass., July 19, 1930, xcv, 189.*—(*G. L. L.*)

Asthma Due to Fungus.—A case is reported of asthma due to a fungus, *Alternaria*, a common genus of the hyphomycetous fungi, belonging to the family *Dematiaceae*. This asthma, in a man aged 37, was associated with a persistent eczema of the hands and feet, and was aggravated by contact with unwashed and musty rags or carpets, clearing up during a sea voyage. Four species of fungi were cultured from a damp cellar where the patient had had several severe attacks while working, and the patient gave a strong reaction to *Alternaria*. It seems probable that his attacks were due to inhalation of fungous spores. Attacks were induced in the patient by the extract of *Alternaria* in powdered form and also by the broth in which it had been grown. —*Asthma Due to A Fungus—Alternaria, J. G. Hopkins, R. W. Benham and B. M. Kesten, J. Am. M. Ass., January 4, 1930, xciv, 6.*—(*G. L. L.*)

Tuberculin Treatment of Asthma.—The subcutaneous injection of increasing doses of Old Tuberculin in the treatment of asthma was used in a group of 36 patients in whom routine methods had failed. A preliminary intracutaneous test was performed on each patient and it was observed that sensitiveness to tuberculin played no part in the production of favorable or unfavorable results. The conclusions are: The treatment is valuable in cases which prove resistant to other therapeutic measures; it can be used in spasmodic asthma in patients of any age and is without harmful effect; the course should be prolonged and repeated if necessary; the results compare favorably with the other therapeutic measures.—*Tuberculin Treatment of Asthma, J. Maxwell, Brit. M. J., May 10, 1930, no. 3618, 854.*—(*M. E. P.*)

Allergy and Cough.—Cough, which often accompanies vasomotor rhinitis as well as bronchospasm, has always been considered secondary to these conditions and never as an independent symptom of allergy. The authors describe three cases in which cough alone, unaccompanied by either rhinitis or wheezing, was due to a hypersensitiveness to, respectively, face powder and feathers, ragweed and feathers, and animal emanations. These cases first of all demonstrate the clinical importance of noting the criteria of allergy. The later occurrence of wheezing in all the cases may be regarded merely as a confirmation of the allergic background. In many instances

an existing hypersensitiveness manifests itself only in the course of an acute infection. Allergic cough has not been emphasized in the literature. When hypersensitiveness forms the background in pulmonary or nasal disease, the recognition and elimination of the offending allergen may be followed by marked amelioration of the disease, even in its advanced stage.—*Studies in Asthma. IX. Cough as a Manifestation of Human Hypersensitiveness, A. Colmes and F. M. Rackemann, J. Am. M. Ass., July 19, 1930, xcv, 192.*—(*G. L. L.*)

Pulmonary Tuberculosis and Anthracosis.—Pliny and later Paracelsus referred to "a mineral or metallic affection which attacks metal and foundry workers, miners, and others who handle these substances, whether silver, gold, aluminum, sulphur, vitriol, lead, copper, iron or mercury. They suffer from asthma, consumption, vomiting." Agricola in 1556 referred to "respiratory diseases in miners," distinguishing those due to dust and those due to vitiated air. Diembroeck later autopsied some miners' lungs and received the impression of cutting into a stony mass. Ramazzini in 1713 described conditions in more detail clinically and Proust distinguished (1) pneumonoconioses of mineral origin, such as (a) siderosis (iron) and (b) chalicosis (silica); (2) pneumonoconioses of animal origin, due to wool, silk, corn, feathers, hair; and (3) pneumonoconioses of vegetable origin, due to tobacco, cotton, cereal grains, etc. Iron, silica and quartz dust certainly exert a traumatic effect on the epithelial cells, and many of the others may set up ulceration. Such scarifications open the way to the entrance of pathogenic agents, such as pneumococci, staphylococci and streptococci, spirillae and even the tubercle bacillus. As to ability to penetrate the lung, only carbon dust has been recognized as having this property. It is often sterile, and is seldom associated with infectious agents. "Black phthisis," or pulmonary anthracosis, was first mentioned in 1827, by Gobert, a Belgian, under the name *asthme des charbonniers*. He desiccated sputum, and recovered from it a powder giving a carbon flame when burned. English physicians next became interested. Christeson, a chemist, confirmed the presence of carbon particles in the lungs at necropsy and found the fumes of lamps an important factor. Crocq, however, in 1862, concluded carbon was harmless to the lungs and even exerted an atrophic effect on tubercles; also, that coal-miners were highly resistant to true phthisis. Later controversies arose over the nature and effects of the pigment. André, Trousseau, Monaret, Villaret and others, held that the black matter in the lungs was not carbon but of haematic origin, resulting from inflammation, a *melanosis*. Numerous experiments followed and it seems to be definitely determined that most of the inhaled dust does not reach the lungs. Much is deposited on the mucosa of the nares, pharynx and larynx. After several hours' exposure to miners' lamps the mocusa takes on a black color. Only a small fraction penetrates the lung, producing an indelible infiltration which may give rise to no symptoms or which may become pathological from extrinsic and intrinsic causes. Among the intrinsic causes are local nasopharyngeal defects, among the extrinsic an abnormally high concentration of dust in the air. *1: Route of Passage:* If the dust particles reach the finer bronchi and alveoli they are engulfed by large cells, "dust cells," whose action would be almost immediate. Both the free and engulfed dust particles then penetrate the pulmonic parenchyma. There many remain implanted in fixed elements, as if tattooed. Others are eliminated by the lymphatics, reaching the hilum lymph nodes, where they are arrested. Some authors, as Vansteenberg and Gryssez, and Herman, following Calmette, believe that much of this pigmentation results from intestinal absorption of the dust, which passes through the thoracic duct with the chyle. Küss, Lobstein, and others vigorously attacked this view and added evidence of aerial transportation of the dust to the lungs. Then Jousset again investigated the nature of the pigment and found its iron content very high. He concluded it was derived from the haematin of the blood, a degenerative product of a hyperaemic reaction. The pigment also is found chiefly around the blood-vessels and not in avascular areas. He regards the famous "dust cells" as scavengers rather than sentinels. Jousset's explanation accounts for the "blackish catarrh" often noted in fibrous tuberculosis, even in mountainous regions, and possibly also for the observations of Corper and Robin, and Willis, who found that inhalations of lamp-black accelerated tuberculosis in guinea pigs. It also accounts for localization of the black in the apices and lymph nodes where tuberculous lesions have produced inflammatory reactions. The question of melanin pigmentation should be reopened. *2: Nature and Properties of Coal Dust:* Most authors have devoted themselves to the dust from lamps or other utensils. Coal dust from the earth, on the other hand, is very different, its particles being generally larger and of more unequal dimensions. Besides certain small amorphous grains are others with a

structure resembling certain lower vegetables, and some containing iron, pyrites and sulphur. In the sputum of anthracotic colliers some particles are as large as 150 or 160 μ in diameter, true foreign bodies in the bronchi; considerably larger than the defending cells. *3: Physiological Factors:* The following unfavorable conditions are met with by coal miners: (1) absence of solar light; (2) deficiency of oxygen; (3) stagnancy of air; (4) toxic atmosphere containing carbonic acid and carbon monoxide, hydrogen sulphide and certain malodorous products of decomposition; (5) chilling by drafts of warm and cold air; and (6) presence of suspended dust,—coal, iron, silica, etc. Independently of the dust the other factors should and do exercise an unfavorable effect on the respiratory tract. Half of the morbidity among miners is due to respiratory diseases, such as pharyngitis, bronchitis, bronchiolitis, generalized or localized alveolitis, pulmonary congestion, bronchopneumonia, pneumonia. It is conceivable that the lungs may be so altered functionally that the cells charged with defense of the alveoli may become enfeebled or devitalized. The cellular elements of alveolar defense consist of (1) anucleated placques protected by thin lamellae, 40 to 100 micra in diameter, and (2) smaller fatty cells, 2 to 15 micra in diameter, with oval nuclei. Their cytoplasm may contain fatty granules. These nucleated fatty cells may detach themselves from the alveolar walls and become "dust cells." *4: Action of Coal Dust on Sensitized Human Respiratory Mechanism:* Gradually the epithelium lining the air-passages undergoes desquamation with loss of ciliary vibration, and cul-de-sacs form into which inhaled dust readily enters. Little by little the dust reaches the alveolar territory and blocks sections of it. Some will be thrown out by coughing and some ingested by phagocytes and transported. Masses of dust, however, accumulate in the alveoli and bronchial ramifications. The reticuloendothelial system is stimulated by its presence and fibrosis develops. Again, dust masses are points of attraction for pulmonary infection. If this is virulent the patient may rapidly succumb to pulmonary oedema or gangrene may develop. Infections of lower grade tend to gradual necrosis and cavitation, with expectoration of foul blackish sputum, "black phthisis." *5: Search for Tubercle Bacilli in Sputum:* Tuberculosis is rarely found among coal-miners, for which various reasons are given. In the first place, the tuberculous and pretuberculous do not usually stand rough work. Of over 10,000 workmen of all ages examined, only 30 presented stethoscopic signs indicative of second- or third-stage tuberculosis and these had not tried work in the bottom of the mine. On the other hand, of 2,250 miners ill over two weeks who were examined at various visits from 1919 to 1923, 877, or 39 per cent, suffered from respiratory affections. In rare cases tubercle bacilli were found in the sputum but most of them showed definite signs of infiltration of the apices, emphysematous bronchitis, pulmonary sclerosis, and some of them cavities. In those in whom repeated examination of the sputum failed to reveal tubercle bacilli the diagnosis was made of "false tuberculosis in miners." Since 1924 all workmen with chronic respiratory affections have been repeatedly examined. About 1 per cent showed definite evidence of true bacillary tuberculosis. This coincides with the investigations of Wedekind and with the low tuberculosis mortality among coal-miners. Wedekind found experimentally that carbon stimulates the reticuloendothelial system and advocates intravenous injections of a fine suspension in the treatment of pulmonary tuberculosis, adding also calcium chloride. Thus he reverts to Crocqu's notion of carbon as an antidote for tubercle bacilli. One recalls the results obtained by injection of gold salts and desires to see further investigations carried out.—*Tuberculose et anthracoses pulmonaires, M. Stassen, Rev. Belge d. l. Tuberc., July-August, 1929, xx, 147.*—(*A. P.*)

Silicosis.—*I: Aetiology* (by Mavrogordato): Phthisis due to dust is a disease of the lungs consequent upon inhalation of certain dust, especially free silica, which seems to attack and excite the reticuloendothelial system. The particles are arrested and removed by phagocytosis, in which the large mononuclear cells or "macrophages" of Metchnikoff play the chief rôle. Silica dust belongs to a group of particulate matter which does not necessarily destroy the cell which absorbs it, neither is it necessarily destroyed itself. This property is also shared by the tubercle bacillus. The result of invasion is the collection of numerous phagocytes into tubercles or pseudotubercles. These phagocytes lose their staining reaction and remain in a state of lowered vitality. The reticuloendothelial system preserves much of its embryonal plasticity and its cells vary in morphology. There is a tendency to transformation into fibrous tissue and a multiplication of fibroblasts. A simple silicosis presents the picture of "anarchic phagocytosis." Most of the dust particles found in the lungs are 5 μ or less in diameter, or about that of the common microörganisms. Two factors are impor-

tant in aetiology: (1) concentration of the dust in the air, and (2) duration of exposure. Continued and prolonged exposure to even low concentrations induces the disease. If fibrosis is the end-result of the arrest of the dust in the lungs, nevertheless it does not constitute the immediate response. Also, if a sufficient quantity of silica is blocked off in the lungs, it will sooner or later give rise to clinical manifestations, but a lung may be invaded without typical silicotic symptoms. The subsequent reaction to experimental tuberculous infection differs in silicotic animals in important respects from the controls. In the disease as encountered in industry, a part of the inhaled silica is retained by "dust cells," which accumulate in pseudotubercles; others pass into the lymphatic system and obstruct it as a river is obstructed by floating driftwood or debris or chunks of ice. The powers of reaction of such a lung toward infection are then altered. Fibrosis of the affected organ is the result of its invasion by the fine dust of simple silicosis. Phthisis due to dust, however, is almost always complicated by secondary invasion by pathogenic microorganisms, especially the tubercle bacillus. It is, then, instructive to speak of "tuberculosilicosis" or of "silicotuberculosis," since simple silicosis does not progress once the dust exposure has ceased. An acute pulmonary infection, however, may attack lungs invaded by silicosis and their powers of resistance to such infections seem to be reduced. Where systems of moistening the air are used to remove dust, conditions are still more favorable for superinfection. Thus, while simple silicosis is a disease of the reticuloendothelial system, "dust phthisis" is a protean disease, whose pathology varies with the respective rôles of the dust and the tuberculous infection. Again, however, the major symptoms may be due to emphysema and failure of the right heart, rather than tuberculosis. When the dust risk is recognized and efforts are made to limit its concentration in the air, clinical modifications of the disease are noted. The tuberculous element comes to predominate, the stigmata of silicosis being subsidiary. With further hygienic improvements the experts in Great Britain and South Africa find that the disease resembles more and more ordinary chronic fibroid phthisis. A supplementary rôle is played by pigmentation. In mines lighted by flame lamps, or in smoky manufacturing towns, a silicotic lung is as black and sooty as that of a coal-miner's, whereas in mines lighted by electricity it is practically free from pigment. Since in many industries water is the only means used to reduce dust, it is probable that the presence of droplets in the air facilitates the entrance of suspended microörganisms and increases the infectious factor. The following factors dominate the incidence of "dust phthisis:" *1:* Concentration of phthisiogenic dust in the air, exposure to infection, temperature, humidity, presence of other dusts, possibly transported, ventilation. *2:* Duration and nature of exposure. *3:* Human factor. Individuals vary in their reactions to dust, and as Haldane says, "man is not a machine." *II: Diagnosis* (by L. C. Irvine, Johannesburg): *1:* This report deals exclusively with silicosis as met with in the gold mines of the Witwaterstrand. The Miners' Phthisis Medical Bureau of Johannesburg has adopted certain criteria based on extensive experience. *2:* Miners' phthisis is the product of two factors, dust and tuberculosis. The initial modifications are due to dust, which predisposes in diverse ways to tuberculous invasion. *3:* The silicotic process begins as a progressive lymphatic fibrosis, gradually developing. *4:* The onset of the malady, from a practical viewpoint, coincides with the development of fibrosis, visible and perceptible in the form of miliary nodules of lungs and pleura. The most striking macroscopic aspect of a confirmed case of simple silicosis is a more or less generalized nodular state symmetrically distributed throughout both lungs; its stages may be practically diagnosed according to the size and number of these nodules. *5:* In most cases of apparently simple silicosis there is early an associated element of tuberculous infection. *6:* Acute infections also occur in silicotic lungs and in turn influence the massive infectious fibrosis. *7:* Latent tuberculous infection probably is present from the onset in most cases. *8:* Most characteristic of silicosis is the generalization of the lesions and the most trustworthy single diagnostic method is roentgenography, which however should be supplemented by careful clinical examination. Judgment in these cases should be rendered by an expert body of diagnosticians who have had experience with the disease in all its aspects. *III: Clinical Aspects of Silicosis* (Boehme): This discussion is based on roentgenographic observations, which, often repeated, afford opportunity for diagnosis of silicosis at its onset and have coincided closely with necropsy findings. Two-thirds of the grave forms of silicosis have an associated tuberculosis and tubercle bacilli are demonstrable during life in 40 per cent of all cases. The appearance of diffuse shadows asymmetrically in silicotic lungs, together with the transformation of the normal heart into a small vertical asthenic one, makes one think of tuberculosis. However, the roentgenographic examination

will not always distinguish between simple silicosis and tuberculosilicosis. Loss of weight, night-sweats, progression of the physical signs, and sometimes erythrocyte sedimentation, permit one to make the diagnosis. Silicosis is very frequent in the Ruhr miners who break rock, while coal miners suffer much less. All workmen exposed to an atmosphere rich in quartz dust should be examined regularly with the X-rays. *IV: Symptomatology of Pneumonoconiosis: Silicosis* (*Thiel*): The most important symptoms are dyspnoea, sense of fatigue, sweating, headache, vertigo, palpitation, and in advanced cases acrocyanosis. There is a tendency to catarrh of the air-passages with cough and sputum, usually scanty. Tuberculosis should always be suspected, even in the absence of tubercle bacilli. As for physical signs, diminished respiratory murmur is most common, less often moist and crepitant râles. There is apt to be dilatation and hypertrophy of the right heart. Emphysema, bronchiectasis or gangrene may complicate the picture. *V: Legislation* (*Collis*): Prevention of this affection requires reporting of occupational cases and regulations tending to minimize the risk. Silicosis itself may lead to total incapacity or death, but is more often interrupted by intercurrent pulmonary tuberculous invasion. It is a disease which requires to be compensated for. Nevertheless, in Great Britain it was for a long time excluded from the list of compensable occupational diseases. Finally in 1918, following the example of South Africa, Great Britain adopted a law applicable to a small group of industries, which were obliged to establish funds for compensation. At first tuberculosis specialists passed on the cases, later a committee of medical experts. The province of Ontario, Canada, included silicosis in its accident-insurance law in 1926 and established a committee of reference to determine the existence and gravity of the cases. Recently Great Britain has extended its law to cover workers in metallic industries, also those working with quartz dust, quartzite, flint and rocks containing 50 per cent or more free silica. Certain states in the United States have enacted legislation, one of the most concrete examples being Wisconsin. *VI: Pneumonoconiosis* (*Sternberg*): The following groups are distinguishable: (1) general allergy combined with asthma; (2) local chromatic changes (anthracosis) or more chemical effects (silicosis, chalicosis); (3) local colloidal effect (amyloses of bronchi); (4) infectious element (tuberculosis, pneumonia, mycosis, psittacosis); (5) possible predisposition toward neoplasms. The physicochemistry of the various dusts is a study of utmost importance.—*La question de la silicose pulmonaire à la IV^e^ réunion de la Commission Internationale pour l'étude des maladies professionelles, Rev. Belge d. l. Tuberc., July–August, 1929, xx, 161.*—(*A.P.*)

Asbestosis.—In a patient with pulmonary asbestosis, three points of special interest were observed, namely, (1) the rapid evolution of the tuberculous process in a patient who had been exposed to asbestos dust; (2) the presence in the lungs of a large number of asbestosis bodies within two years of the first exposure to the dust; and (3) the presence in the lungs of asbestos fibres and of various stages in the formation of typical asbestosis bodies.—*A Case of Pulmonary Asbestosis: Death from Tuberculosis Two Years after First Exposure to the Dust, W. B. Wood and D. S. Page, Tubercle, January, 1930, xi, 147.*—(*W. I. W.*)

Asbestosis.—Asbestos fibres, when injected into the body, act as a benign irritant, producing granulation tissue. This granulation tissue contains many asbestosis giant cells, indicating presumably an attempt to destroy the asbestosis fibres by phagocytosis. Connective tissue is formed in due course, but the giant cells persist. The asbestosis giant cell is readily distinguished from the giant cell in tuberculosis. Asbestosis bodies were not found. Asbestos injected repeatedly intravenously appeared to have no toxic affects on distant tissue.—*The Reaction of Tissues to the Asbestos Fibre, with Reference to Pulmonary Asbestosis, S. R. Gloyne, Tubercle, January, 1930, xi, 151.*—(*W. I. W.*)

Pulmonary Siderosis.—Although siderosis was the first of the dust diseases to be described, it has received less attention than anthracosis and silicosis. Among metal-workers and iron-miners a large proportion show respiratory symptoms, though these have been reduced by the use of devices for diminishing dust. Langguth has found 7.9 per cent of the dry weight of a siderotic lung to be iron oxide. It has not been possible to produce siderosis in animals; hence, human material offers the main field for investigation. The disease occurs in two forms, (1) with a red type of lung, due to absorption of iron oxide dust in iron mines, and (2) with a black type of lung, due to absorption of the dust produced in metal-polishing or grinding when fine particles of iron and silicates from the grind stones are thrown into the air. Clinical records and detailed autopsy findings are given of a case of each type. The body of a former metal-polisher who had died of

tuberculous bronchopneumonia showed six large brown substernal nodes, and in the apices of both lungs there were firm masses of solid grayish and grayish-brown tissue, in which pearly gray streaks could be seen. In the lower lobes there was similar tissue, but of nodular form. In the lower lobes there were also many grayish-white gelatinous patches. The tracheobronchial lymph nodes were large and brownish. The dark nodules from the lower lobe on microscopic section showed a central arteriole surrounded by large pigment-filled cells. The lining cells of the smaller bronchi showed granules of iron pigment. There were also areas of confluent tubercles. The anatomical diagnosis included pulmonary siderosis of the black type, siderosis of the tracheobronchial, anterior mediastinal and peripancreatic lymph nodes, tuberculous bronchopneumonia, and tuberculosis of the tracheobronchial lymph nodes. The second case was that of a former miner who died in great dyspnoea. On autopsy there were several large, firm, very brown substernal lymph nodes. Both upper lobes contained hard deep reddish-brown masses, and extending down through the lungs there were similar but smaller areas. The tracheobronchial lymph nodes were enlarged, firm, brown and discrete. Microscopic sections of the brown mass in the upper lobe showed only masses of brown pigment; the smaller nodules in the base showed central vessels surrounded by concentric fibrous tissue cells in the meshes of which were numerous large cells distended by iron pigment. The anatomical diagnosis included pulmonary siderosis of the red variety, siderosis of the tracheobronchial, substernal, and peripancreatic lymph nodes, and siderotic deposits in the capsule of the spleen. There is a discussion of the fate of the iron entering the body through the respiratory passages. Iron-bearing macrophages probably enter the alveoli and are expectorated. It is therefore possible in many instances to detect iron in suspected cases of siderosis by the deep blue color developing after acidifying the sputum with hydrochloric acid and adding potassium ferrocyanide. The pigment which is carried to the lymph nodes is either deposited there or is carried through and reaches the blood-stream, probably by way of the thoracic duct. From the blood-stream it is deposited in the cells of the reticuloendothelial system. The fate of this transportable iron is discussed fully. It is of note that despite the large amount of circulating iron both patients had anaemia. Where the accumulations of iron were greatest, the amounts present were 8.9 and 7.9 per cent of the moist weight of the lungs.—*Pulmonary Siderosis, M. G. Bohrod, Arch. Path., August, 1930, x, 179.—(R. D.)*

Deformity of Thorax.—Deformity of the thoracic cage follows a marked grade of scoliosis or kyphosis from any cause. Such deformity may or may not involve the spine. When a marked grade of secondary deformity of the spine does develop it may produce markedly increased deformity of the ribs and other bony structures. The diseases of the pleurae or lungs which produce thoracic deformities are chiefly the chronic inflammatory processes which result in extensive scar-tissue contraction. The type of change in the bony framework varies in part as a result of the direction of the pull exerted by the scar-tissue and in part as a result of the relative mobility of the structures subject to traction, such as the ribs, spine, mediastinum and diaphragm. Some cases of empyema will produce changes only in the ribs; others result in a marked scoliosis. Chronic pulmonary fibrosis may effect a marked retraction of the chest-wall, and in others a displacement of the mediastinal structures to the extent of complete dextrocardia without noticeable changes in the ribs or spine. The main factors in the development of a marked grade of empyematous deformity are chronicity and lability of the structures. Therefore deformity is most apt to occur in cases in which the empyema begins in childhood and in which the suppuration is of long duration. The empyema may be of the ordinary pyogenic type or it may be tuberculous with or without a secondary pyogenic infection. The rib deformity in some chronic empyemata may be extreme, the ribs lying in direct contact, overlapping, or fused into a bony plate, and being usually of increased density. The deformity of the spine is usually a scoliosis, the main dorsal concavity of the spine being usually toward the affected side. In extreme cases of empyematous scoliosis the downward and inward deflection of the ribs on the affected side is so great that the pleural cavity on that side is greatly reduced in volume. In primary scoliosis, on the contrary, the ribs on the side of vertebral concavity are elevated above the normal, resulting in an increase in volume of the pleural cavity on that side. In chronic fibroid phthisis the deformity is usually chiefly of the ribs, with a contraction of the hemithorax, the intercostal spaces being narrowed, and the direction of the ribs more oblique. If scar-tissue retraction of pleura and lung is the true explanation of deformities of the aetiological types under consideration, release of such scar-tissue traction should constitute a rational method of treatment. The author

has performed resection of the parietal pleura and a pulmonary decortication in a number of cases with gratifying results, the deformity being in part corrected, and the pulmonary function increased. In case of cicatricial contraction of a diseased lung resulting in mediastinal displacement, mobilizing the chest-wall, by excising segments of the ribs posteriorly after the method of Sauerbruch, releases this traction on the mediastinum and promotes the healing process in the lung. In cases of scoliosis associated with fibroid phthisis with the typical concavity toward the diseased lung, a posterior thoracoplasty often serves to correct the scoliosis. Occasionally an overcorrection is obtained, resulting in a mild curvature with its concavity toward the sound side. In explanation it must be assumed that the scoliosis is secondary to a force exerted against the spine by the ribs. Normally this force is equal on the two sides. In the case of a fibrotic lung producing a narrowing of the intercostal spaces, the lateral pressure of the ribs on this side is increased; hence the curvature toward this side. When the ribs on this side are resected, the unopposed normal pressure from the opposite side corrects the deformity and in case of a very mobile spine may result in a slight overcorrection.—*Deformity of the Thorax Secondary to Pleural or Pulmonary Disease, C. A. Hedblom, J. Am. M. Ass., January 18, 1930, xciv, 162.*—(*G. L. L.*)

Ileocaecal Gurgling in Chest-Wall.—The author reports 5 cases from the literature and 2 personal observations in which the intact and noninflamed skin of the chest exhibited a fluctuating tumor with gurgles removed by pressure. Following cough or pressure the tumor again appeared. The gurgling or crepitation is accounted for by the presence of air and fluid and is believed to be due to a communication between a subcutaneous fistula and the lung. As a rule it depends upon a perforated cavity with empyema in which the pus finds its way by burrowing under the skin.—*Das Phänomen des ileo-cöcalen Gurrens an der vorderen Brustwant, W. Feldmann, Zeitschr. f. Tuberk., 1929, liv, 139.*—(*H. J. C.*)

Diseases of Upper Respiratory Tract.—Roentgenological studies of the chest of infants and young children should always include the neck, in order to detect possible obstructive lesions in the upper respiratory tract that may explain, in part or entirely, suspected intrathoracic conditions. In older children and adults inclusion of the upper tract is necessary in certain groups of cases. As the subject of the examination of the neck is a broad one, the findings in only the very young are discussed at this time. The oesophagus is not included. The literature on this subject is very meagre. The basis for the detection of upper-respiratory conditions is complex. The soft tissues of the neck surround a more or less rigid air-containing passage,—the pharynx, larynx and trachea, which can be visibly encroached upon; a collapsed potential space,—the oesophagus, can be filled by opaque media; the dense vertebrae occupy a fixed position for estimating displacements and making measurements; clear views are easily obtainable of the intrathoracic trachea and its relations; and certain phenomena observable in the chest may be associated with or due to obstruction in the upper air passages. A uniform and comprehensive technique is essential and is described. A study of the normal is necessary as a basis for the diagnosis of pathological conditions. The following structures are easily seen in roentgenograms of the neck at ages when their detection is important: The posterior border of the tongue, uvula, pharyngeal cavity and its walls, epiglottis, aryepiglottic folds, pyriform sinuses, laryngeal vestibule, ventricular and vocal folds, laryngeal ventricles and the lower limits of the laryngeal cavity. The following pathological conditions amenable to roentgenological diagnosis are discussed in detail: Streptococcic laryngitis, acute diphtheria, postdiphtheritic and other forms of stenosis, tuberculous adenitis with calcifications, retropharyngeal and retrotracheal abscess, encroachments on the nasopharynx, foreign bodies, and the thymus. The stand is taken that thymic death is due largely to obstructive tracheostenosis, but is usually assisted by other factors aiding in upper respiratory obstruction. It is realized that much of the diagnosis of enlarged thymus is based upon inadequate examinations. A comprehensive technique is described. The only definite or reliable signs of an enlarged or potentially obstructive thymus are narrowing of the trachea at the upper thoracic outlet on inspiration, too great a collapse on expiration or buckling at either phase, as shown by the lateral view, or lateral deviation of the trachea shown in the sagittal view. The preponderant thymus of infancy and early childhood may be blamed for obstructive phenomena for which it is not responsible or which aid in the obstruction or cause a persistence of the symptoms after subsidence of the gland by treatment. In this connection, the examiner must have in mind the following: Foreign bodies, retropharyngeal abscess, tracheal and laryngeal stenoses, recurrent laryngeal paralysis from the thymus, the relaxed soft tissues of the pharynx and larynx in the young, asthma,

whooping-cough, meningitis, and congenital heart conditions.—*Roentgenologic Diagnosis of Diseases of the Upper Respiratory Tract in Children, H. K. Pancoast and E. P. Pendergrass, Am. J. Roentg. & Rad. Therapy, March, 1930, xxiii, 241.*—(*W. I. W.*)

Frequent Chest Colds.—There is considerable variability in the occurrence of chest colds in those who are very susceptible, and this seems to be an individual characteristic rather than one due to dependence on extraneous conditions. Bacteriological examination of the sputum at the time of a chest cold reveals variability among the varieties of streptococcus, which is the organism most constantly isolated. For these reasons, and since a vaccine consisting of the most prevalent varieties of streptococci seems frequently to prevent and often to benefit a cold in the chest, it is fair to assume that streptococci play a part in the cause of colds.—*Frequent Chest Colds, I. C. Walker and J. Adkinson, Arch. Int. Med., July, 1930, xlvi, 1.*—(*W. I. W.*)

Aetiology of Psittacosis.—In three parrots which had apparently been the source of severe infections in human beings, a filterable virus was demonstrated. With it, the disease could be caused serially in several generations. In the pericardial fluid of infected animals, very minute and very numerous coccoid bodies could be demonstrated. The same elements were seen in virus-containing filtrates. It is suggested that these elements represent the aetiological agent of psittacosis.—*Die Ätiologie der Psittakosis, W. Levinthal, Klin. Wchnschr., April 5, 1930, ix, 654.*—(*M. P.*)

Psittacosis.—In May, 1929, three patients were observed in Graz whose final diagnosis was psittacosis. All three came from the same household, in which parrots were kept. Nine parrots had died with a suspicious symptomatology. The clinical course in the first 2 cases was that of severe bronchitis, with multiple lobular pneumonic foci. The general symptoms resembled those of typhoid fever. All bacteriological and serological tests on the blood were negative. The third patient was a man, sixty-eight years old, who had suffered from chronic myocarditis for several years. He developed pneumonia of the left lower lobe and bronchitis in the rest of the lung. He was delirious from the first day of his disease and died on the fourth day. Both clinical and epidemiological observations made it most likely that all three patients had suffered from psittacosis.—*Ueber drei Fälle von Psittacosis, J. Widowitz, Wien. klin. Wchnschr., February 13, 1930, xliii, 195.*—(*M. P.*)

Psittacosis.—Five cases of psittacosis are reported. These patients gave a history of having been in contact with a parrot suffering from this disease. The outstanding features of the disease were (a) the high infectivity of the disease, (b) the absence of a single case of infection from a human being, and (c) the extreme severity of the illness. Two of the patients had symptoms of typhoid fever, three had pulmonary symptoms, but none showed consolidations of the lung.—*An Outbreak of Psittacosis, M. C. Radford, Brit. M. J., February 22, 1930, no. 3607, 333.*—(*W. I. W.*)

Psittacosis.—Twenty-seven cases of psittacosis are reported. The onset in most of these patients was sudden. The initial symptoms were headache, malaise, shivering, pains in all the limbs, backache, abdominal pain, anorexia, nausea, vomiting, sore throat, sweating, and epistaxis. Cough developed usually toward the end of the first week. It was usually dry, but in several of the cases there was a considerable quantity of mucopurulent sputum. Of these 27 patients, 25 were apparently infected through green Amazon parrots from South America. The mortality in this series was 20 per cent. The blood-agglutination tests were negative. A filterable virus was isolated from parrots which had died of psittacosis and which had been responsible for cases of the disease in man. The changes found at necropsy were those of a general infection or bacteriaemia, associated with a peculiar inflammation of the lungs.—*A Study of Psittacosis, R. Hutchinson, R. A. Rowlands and S. L. Simpson, Brit. M. J., April 5, 1930, no. 3613, 633.*—(*W. I. W.*)

Psittacosis in Vienna.—In August, 1929, an Italian, forty-three years of age, was observed and diagnosed as having psittacosis. This patient had been in close contact with a large number of Brazilian parrots, many of which had died. Three other members of his family had been sick, approximately at the same time, with similar symptoms. On the basis of these observations, the disease, psittacosis, is discussed. Finkler drew attention to certain atypical pneumonias and the presence of parrots in the same households. In 1887 he observed 6 such patients. Dujarden and Beaumetz, in 1892, assumed that parrots transmitted the influenza virus. Nocard reported, in 1893, the discovery of the bacillus which he claimed to be the specific cause of psittacosis. Nocard's bacillus is probably identical with the *bacillus para-*

typhosus enteritidis Breslau. In a few single cases it was possible to isolate this organism from diseased parrots, but in all later epidemics it has never been possible to demonstrate its presence in human lesions. Various other microörganisms have been found in association with the disease. The aetiological rôle of Nocard's bacillus is highly doubtful, but the fact that psittacosis is transmitted by parrots rests on sound epidemiological observations. In the human being the disease seems to have an incubation period of about ten days. It begins suddenly, with fever, prostration and loss of appetite, but without herpes. Cerebral symptoms, such as headache, clouding of consciousness, and delirium are frequent. The temperature lasts, usually, fifteen to twenty days. Physical examination reveals the symptoms of a capillary bronchitis, frequently with the development of lobular pneumonic foci. Pleural effusions are frequent. The sputum is scanty, mucous, and sometimes sanguineous. The cardiovascular system shows signs of toxic impairment. The pulse is small and soft. The spleen is usually enlarged. Gastrointestinal disturbances are frequent. The clinical picture resembles very much a typhoid-pneumonia. The mortality is as high as 45 per cent. At necropsy, the spleen was not always found to be enlarged. The pneumonic foci are smooth on cut surface, dark red, moist, and glistening. No exudate can be expressed from the infiltrated portions. Histologically, the foci contain very little fibrin. The exudate is both intra- and interalveolar. The diagnosis cannot rest on the bacteriological findings; it must be made from clinical and epidemiological observations.—*Ueber eine kleine Endemie von Psittacosis in Wien (August, 1929), O. Weltmann, Wien. klin. Wchnschr., February 13, 1930, xliii, 193.*—(*M. P.*)

Bacterial Flora of Influenza.—The flora in the sputa of patients acutely ill with influenza were identical in containing two predominating types of bacteria: green-producing streptococci and gram-negative diplococci. The streptococci of these patients were pathogenic for animals. These streptococci produced a toxin. Skin tests with this toxin suggested a means of determining susceptible persons. Vaccines made from these streptococci seemed to have the power of immunizing against the influenza infection. The green-producing streptococci for the most part reacted similarly in fermentation tests and bile-solubility tests. These streptococci showed relationship in cross-agglutination experiments.—*The Influenza Epidemic of 1928, E. F. Traut and R. D. Herrold, Arch. Int. Med., March, 1930, xxxv, 412.*—(*W. I. W.*)

Unresolved Pneumonia.—*Case Reports:* Seven patients with unresolved pneumonia were treated with roentgen irradiation of the chest. Four cleared up entirely, two definitely improved, and one remained unchanged. The results in these few patients have been prompt and gratifying with no untoward effects.—*Roentgen-Irradiation in Unresolved Pneumonia, E. A. Merritt and E. M. McPeak, Am. J. Roentgenol. & Rad. Ther., January, 1930, xxiii, 45.*—(*W. I. W.*)

Carbon Dioxide in Pneumonia.—After surgical operations, massive lobar or lobular atelectasis of the lung is a rather frequent occurrence, and is the condition from which postoperative pneumonia develops. This apneumatosis is prevented and relieved and the risk of pneumonia is eliminated by the inhalation of carbon dioxide. The inhalation of 5 per cent carbon dioxide in oxygen, which is now the standard treatment for carbon-monoxide asphyxia, is also an effective preventive of postasphyxial pneumonia. In pneumonia, it is the blocking of the lung air-ways, bronchi or bronchioli, by plugs of thick and sticky secretion which is the critical morbidic factor producing atelectasis and the conditions characteristic of an undrained infection. Atelectasis, induced experimentally in dogs by blocking a bronchus, is quickly cleared up and the lung is redistended by induced deep breathing, by inhalation of carbon dioxide in proper concentration. Pneumonia, induced in dogs by insufflation of a virulent culture of pneumococci, is generally overcome, the lung is redistended, and the animal is restored to health by inhalation of carbon dioxide sufficient to cause deep breathing, and continued until the pneumonic area is cleared. Success with this therapy for the relief of pneumonia in patients depends on administering the inhalation as early as possible. If pneumonia is thus treated early enough, it appears probable that the results may be as effective as those already obtained in postoperative and postasphyxial pneumonia.—*The Treatment of Pneumonia by Inhalation of Carbon Dioxide, Y. Henderson, H. W. Haggard, P. N. Coryllos and G. G. Birnbaum, Arch. Int. Med., January, 1930, xlv, 72.*—(*W. I. W.*)

Pulmonary Tuberculosis and Pneumonia.—The occurrence of lobar pneumonia with pulmonary tuberculosis is relatively rare, and the latter is not particularly unfavorably influenced by the acute disease. Although this is a well-known observation

the explanation has not been satisfactory. The author believes that the explanation lies in certain immunobiological factors.—*Lungentuberkulose und Lungenentzündung. Zugleich ein Beitrag zur Tuberkuloseallergie, Rehberg, Beitr. z. Klin. d. Tuberk., 1929, lxxi, 6-10.*—(*H. J. C.*)

Glanders of Lung.—A case of glanders of the lung simulating pulmonary tuberculosis is reported. Because tubercle bacilli could not be demonstrated in the sputum, a guinea pig was inoculated. The organism isolated from the guinea pig was reported to be the glanders bacillus.—*A Case of Glanders of the Lungs, Simulating Pulmonary Tuberculosis, E. H. A. Pask, Tubercle, March, 1930, xi, 257.*—(*W. I. W.*)

Dengue Fever and Pulmonary Tuberculosis.—The author reports upon the effect of dengue fever in an epidemic in Greece in 35 cases of pulmonary tuberculosis. In spite of the fact that dengue causes a decided weakness and diminution in bodily resistance, the effect on pulmonary tuberculosis was not always unfavorable. In healed cases and those with pneumothorax in which a good collapse of the diseased lung occurred, as well as in the majority of cases of latent type, no harm was observed. In the remainder of the cases the detrimental effect was only transient and occurred only in the more severe types. Only in the profoundly toxic and progressive cases was the harm serious. Dengue fever infection can be a causative factor, however, in the occurrence of pulmonary tuberculosis.—*Denguefieber und Lungentuberkulose, B. Papanikolau, Zeitschr. f. Tuberk., 1929, liii, 107.*—(*H. J. C.*)

Emphysema.—The object of this study was to determine whether obstruction of the air-passages may produce emphysema. Numerous methods were used in producing obstruction at various levels of the respiratory tracts of dogs. Long-continued high-grade obstruction of the air-passages of dogs was insufficient to produce a pathological picture simulating vesicular emphysema in man.—*The Etiology of Emphysema, L. M. Loeb, Arch. Int. Med., March, 1930, xxxv, 464.*—(*W. I. W.*)

Spontaneous Pneumothorax and Emphysema.—Tuberculosis plays a predominating rôle in the origin of spontaneous pneumothorax, being the cause in about 80 to 90 per cent of the cases. In addition, pulmonary gangrene, putrid bronchiectasis, pulmonary infarcts and tumors can cause pneumothorax, and influenza as well as rare cases of abdominal disease can be the cause. In phthisis the pneumothorax can develop from a rupture of a subpleural focus or a cavity. In disseminated forms of tuberculosis there can be a tear in a spot of vicarious emphysema. While spontaneous pneumothorax is a bad sign in phthisis there can occur ruptures of the lung in previously healthy individuals but these heal within 6 to 8 weeks. That spontaneous pneumothorax can result in a case of genuine emphysema is evident from a case reported by the author.—*Spontanpneumothorax und Emphysem, H. Lichtenstein, Beitr. z. Klin. d. Tuberk., 1929, lxxi, 183.*—(*H. J. C.*)

Enlargement of Mediastinal Lymph Nodes in Adults.—The diagnosis of this condition is extremely difficult, and no method makes the diagnosis quite certain. For obvious reasons, in many cases, the condition cannot be detected by roentgenological examination. The fact that the enlarged lymph nodes may cause definite pressure symptoms on the surrounding structures, including nerves and blood-vessels, must be borne in mind as substantial evidence. Bronchophony over the *manubrium stern* is suggested as a reliable diagnostic sign. In combination with the other well-known procedures, the correct diagnosis can frequently be made. Enlarged mediastinal lymph nodes are, in some patients, the first objective symptoms of an active tuberculous infection.—*Ueber die Vergrösserung der mediastinalen Lymphdrüsen bei Erwachsenen, L. Vajda, Wien. klin. Wchnschr., November 14, 1929, xlii, 1473.*—(*M. P.*)

Bronchial Obstruction.—From experimental and clinical investigation, atelectasis, postoperative pneumonitis and lobar pneumonia are shown to be produced by the same mechanism. The identity in pathogenesis of these morbid conditions of the lung is proved by the gross and microscopic appearance, their bacteriology, their circulatory disturbances, their evolution, and the modifications in the pulmonary gaseous exchanges occurring in them. Between them, there are (in degree only) differences in the local and general symptoms, roentgenographic signs, and bronchoscopic findings. Lobar pneumonia is a local pulmonary infection with general manifestations. The management of the local infection is the most important part of the treatment. Pneumonia is due to bronchial obstruction. Any interference with the free drainage of the bronchial tree will cause lesions, the nature, extent, importance, and evolution of which will depend upon the degree and duration of the obstruction, and nature and virulence of the

microbes present. The rational treatment of these conditions should be based upon their aetiology and, as in every other obstructive lesion, should aim to relieve the obstruction and reëstablish free drainage of the bronchial tree. Lobar pneumonia is a pneumococcic bronchial obstruction, with a combined atelectasis and pneumococcic cellulitis of the corresponding pulmonary area. Treatment should be directed, not only against the bacteriaemia, but chiefly against the local lesion. Carbon-dioxide inhalation in 5 to 10 per cent concentration seems to possess a specific action against pneumococcic pulmonary infections and, according to experimental and clinical data, could be used both as a preventive and curative agent.—*Bronchial Obstruction, P. N. Coryllos and G. L. Birnbaum, Am. J. Roentgenol. & Rad. Therapy, November, 1929, xxii, 401.* —*(W. I. W.)*

Massive Collapse of Lung.—Though this condition was studied and much discussed in the first half of the nineteenth century, the first description of it as it is now understood has been credited to Pasteur's paper of 1890. Later he so perfected the diagnostic criteria that little has been added. Cumulative evidence, both clinical and experimental, favors bronchial obstruction as the cause, and mucous plugs in the bronchi as the most constant factor. Wilson and Habliston recently described the occurrence of high negative intrapleural pressure and demonstrated the therapeutic value of artificial pneumothorax. A history of bronchitis, haemoptysis, or foreign-body aspiration may be obtained. Trauma and surgery are predisposing factors, and all intrathoracic processes which might occlude a bronchus must be considered. The first symptoms are usually fever and rise in pulse and respiratory rate. Soon chest pain and respiratory distress occur. Physical signs vary with the extent and degree of collapse, and the presence or absence of compensatory emphysema. Typically, one sees an anxious expression, rapid breathing and cyanosis. The side affected is relatively immobile with retraction of the chest-wall. Expansion of the contralateral side is exaggerated and the chest-wall prominent. The trachea and apex-beat are displaced toward the affected side. Vocal fremitus is decreased or absent over the collapsed lung, and resonance is lost. Auscultatory signs vary. The X-ray confirms the physical signs. The onset in spontaneous pneumothorax may be similar. An understanding of the principles underlying both types of collapse is therefore necessary. In massive collapse the lung is airless because of bronchial obstruction and absorption of air, with a resultant negative intrapleural pressure, and the mediastinal contents are displaced toward the affected side. In pneumothorax, the lung is collapsed because of pressure of air in the pleural space. The mediastinal structures are pushed away from the affected side, and the diaphragm is displaced downward. Gwyn recognized massive collapse as an occasional complication of pneumonia. Consolidation in pneumonia is differentiated by the absence of cardiac displacement and by the characteristic systemic signs. Pulmonary embolism and acute heart failure are confused with massive collapse only because of the similarity of onset. Pleural effusion is differentiated by history and the characteristic physical signs. Treatment of massive collapse must eventuate in removal of the bronchial obstruction. Sante's method of rolling the patient back and forth on the unaffected side should be tried as first aid. If this fails, artificial pneumothorax should be induced. This facilitates bronchoscopy, should it be needed. Pneumothorax relieves the distressing symptoms. A case is reported in which bronchitis and an abdominal abscess were associated at the onset of illness. The abscess was on the right, and about four days after its drainage, massive collapse of the right lung occurred. X-rays show the rapid reëxpansion and clearing of the collapsed lung under artificial pneumothorax treatment.—*Massive Collapse (Atelectasis) of the Lung, with a Case Report, L. J. Moorman, Ann. Int. Med., April, 1930, iii, 1024.*—*(R. M. F.)*

Massive Collapse of Lung.—The typical case of lobar collapse of a lung has a sudden onset of sharp pain in the side; difficulty in breathing; cyanosis; moderate rise in temperature and pulse; tendency for the patient to lie on the affected side; retraction and immobilization of the affected side of the chest; dulness, absent breath-sounds and tactile vocal fremitus over the collapsed portion of lung; and slight cough, with expectoration of tenacious mucus. The X-ray picture shows a dense homogeneous shadow over the site of the lesion; displacement of the mediastinum and its contents toward the affected side; reduction in width of the chest cavity on the affected side with narrowing of the costal interspaces; elevation and fixation of the leaflet of the diaphragm on the affected side; and compensatory emphysema of the unaffected lung. It occurs after operations and trauma, and is sometimes associated with pneumonia and tuberculosis. Active lobar collapse must be differentiated from massive collapse of the lung in cases of pleural effusion, pneumo-

thorax, and bronchial obstruction by new growth or foreign body. In the latter cases the collapse is a gradual process and the symptoms are rarely distressing. Many theories have been advanced for the explanation of this phenomenen, but the weight of evidence is in favor of the theory that spasm of the bronchial musculature is the essential feature of active lobar collapse. There is a reflex disturbance of the sympathetic and vagus innervation of the lungs. First there is dilatation of the bronchi with cessation of the wave-like movement and a consequent accumulation of mucus, due to overaction of the sympathetic nerve. Then the vagus may assert itself too strongly and cause a sudden spasm of the musculature which empties the lung tissue of its air content. When the walls of the alveoli and terminal bronchioles are brought into apposition, the molecular adhesion effectively prevents the reëntry of air. Gradually the balance between the vagus and sympathetic is restored and air reënters the alveoli. The termination of these cases is almost always favorable. —*Massive Collapse of the Lung, J. M. W. Morison, Brit. M. J., August 16, 1930, no. 3632, 237.*—(*M. E. P.*)

Bronchoscopy in Atelectasis.—Atelectasis accounts for a majority of all postoperative pulmonary complications. By means of bronchoscopy, some of the postoperative changes in atelectasis have been observed. It appears that the process of anaesthetizing the pharynx and cords and the introduction of the bronchoscope itself may free otherwise firmly fixed bronchial plugs. The observation of the almost complete occlusion of a main bronchus by an oedematous swelling suggests a process akin to angioneurotic oedema of other tissues. Carbon-dioxide inhalations, when observed through the bronchoscope, were seen to (1) increase the rate and depth of respiration, (2) produce violent movements in the tracheobronchial tree and alterations in the shape of the lumina of its branches, thereby tending to free adherent mucus, and (3) induce a distinct blanching of the mucous membranes of the trachea and bronchi.—*Bronchoscopic Observations in Postoperative Atelectasis: Action of Carbon Dioxide, A. L. Brown, J. Am. M. Ass., July 12, 1930, xcv, 100.*—(*G. L. L.*)

Atelectatic Bronchiectasis.—A series of cases are reported in which the roentgenograms of the chest showed a triangular shadow at the base of the lung. These patients gave a history of raising much sputum, or dated their cough from their recovery from pneumonia. The injection of lipiodol demonstrated bronchiectasis within the shadow. The triangular shadow lay in the recess formed by the spine and the ribs and the angle of the diaphragm. This area is shielded from the more forcible respiratory movements and is a location where one would expect to find a residual atelectasis. The primary condition in these patients has been described as an atelectasis and bronchiectasis. This type of pathological change may account for some cases of continual attacks of haemoptysis with no definite signs of tuberculosis.—*Atelectatic Bronchiectasis, A. J. S. Pinchin and H. V. Morlock, Brit. M. J., January 4, 1930, no. 3600, 12.*—(*W. I. W.*)

Haemorrhagic Dry Bronchiectasis.—Patients presenting the symptom of haemoptysis, when the physical signs are inconclusive, and X-ray studies show no definite evidence of pulmonary tuberculosis, should be subjected to lipiodol injection of the bronchial tree, in order to exclude the possibility of apical or basal dry bronchiectasis. The authors have seen 15 cases during the last two years and describe *9* of them.—*Haemorrhagic Dry Bronchiectasis, A. J. S. Pinchin and H. V. Morlock, Brit. M. J., August 30, 1930, no. 3634, 315.*—(*M. E. P.*)

Emetine in Treatment of Bronchiectasis.—In the treatment of bronchiectasis the following factors are used chiefly: Climate, postural drainage, expectorants, injection of ethereal oils, respiratory exercises, dry diet in combination with the injection of hypertonic salt solution and atropine, autogenous vaccines, and, in unilateral lesions, collapse therapy. Recently, Sternberg advocated the use of emetine in combination with neosalvarsan and trypaflavine for the treatment of pulmonary suppuration. During 15 months a number of children with bronchiectasis and chronic bronchitis were treated with daily subcutaneous injection of 0.02 to 0.03 gm. of emetine hydrochloride in a 10 per cent solution. After ten or twelve days the injections were stopped, and after another period of about ten days the patients received, at daily intervals, salvarsan or transpulmine. The results, after a year's observation, were quite satisfactory; the symptoms and expectoration disappeared completely or were greatly diminished. Since the clinical diagnosis of bronchiectasis is usually made only after the lesion is far advanced it is advocated to treat with emetine in combination with salvarsan or transpulmine, all those diseases which are known to cause or prepare the ground for bronchiectasis.—*Die*

Behandlung der Bronchiektasien im Kindesalter, F. Basch, Wien. klin. Wchnschr., March 20, 1930, xliii, 361.—(M. P.)

Pulmonary Infarction.—Twenty-five cases of pulmonary infarction, in which the clinical and roentgenological findings were carefully studied, are reported. In 17 cases the infarction took place following a surgical operation and in six the accident occurred in patients suffering with cardiac decompensation. Seventy-two per cent of these patients had definite haemoptysis. It occurred in 17 of the postoperative cases and in 5 of 6 of the cardiac cases. Pleuritic pain was present in 76 per cent of the cases. With the clinical data, the nature of the shadows appearing on the films may usually be accurately determined. Infiltration at the bases, especially at the right base associated with thickened pleura are suggestive of pulmonary infarction.—*A Clinical and Roentgenologic Consideration of Pulmonary Infarction, B. R. Kirklin and L. S. Faust, Am. J. Roentg. & Rad. Ther., March, 1930, xxiii, 265.—(W. I. W.)*

Abscess of Lung.—Obstructive atelectasis is a common observation in otherwise healthy albino rats. The obstruction is frequently the result of the accumulation of mucus in the form of a plug completely occluding the bronchial lumen. Microorganisms are frequently obtained from the lungs of healthy animals. On the basis of these observations, it is probable that the sequence of events in the pathogenesis of pulmonary disease in the white rat is the entrance of organisms into the lungs, an obstructive atelectasis followed by the growth of organisms distal to the point of occlusion and the development of pulmonary suppuration. The established suppurative process may extend to other parts of the lung by a repetition of these changes or by direct extension to adjacent structures.—*Atelectasis in the Pathogenesis of Abscess of the Lung, T. S. Moise and A. H. Smith, Arch. Int. Med., January, 1930, xlv, 92.—(W. I. W.)*

Abscess and Gangrene of Lung.—It has been much discussed recently whether artificial pneumothorax is indicated in patients with pulmonary abscess. The routine treatment of pulmonary abscess and gangrene in the Third Medical Department of the Wilhelminenspital in Vienna is as follows: First, intravenous injections of neosalvarsan are given. If temperature and sputum are definitely reduced during the first week, this treatment is continued until recovery. Out of 13 patients which were treated in such a way, five were discharged as completely cured, seven were improved, and one patient died within twenty-four hours after admission. If salvarsan treatment does not bring any relief within the first ten days, artificial pneumothorax is induced and salvarsan injections are continued. Out of 13 patients treated by this method, seven were completely cured, four were improved, and two died. If a rupture of an abscess-cavity occurs, drainage of the pleural space is necessary immediately. If pneumothorax cannot be induced, operative procedures are indicated.—*Zur Behandlung des Lungenabszesses und der Lungengangrän, W. Neumann, Wien. klin. Wchnschr., February 27, 1930, xliii, 261.—(M. P.)*

Fusospirochaetal Disease of Lungs.—Vincent's angina, or fusospirochaetal infection of the throat, is a relatively common and widespread disease, and pulmonary complications due to these fusospirochaetal organisms are not infrequent. The complicating pulmonary lesions often confused with pulmonary tuberculosis may take the form of pulmonary gangrene, pulmonary abscess, ulceration of the bronchi, lobar pneumonia, bronchopneumonia and empyema. Many of these patients recover spontaneously, but in some there is a fatal termination. It seems advisable to treat all cases in which fusospirochaetal organisms can be demonstrated in the pulmonary sputum with either neoarsphenamine or sulpharsphenamine. To obtain the best results the diagnosis should be made and the arsenic given before there is any appreciable destruction of pulmonary tissue. A report is made of 3 cases of pulmonary fusospirochaetal disease, that developed following contact with cases of Vincent's angina. In two cases primary infections of the pharynx subsequently extended into the lungs, and the third case was primary in the lungs, developing four days after contact. Treatment with sulpharsphenamine was begun on the third, fourth and fifth days, respectively, of the illness, and in all 3 cases recovery was prompt.—*Relation of Vincent's Angina to Fusospirochetal Disease of Lungs, D. T. Smith, J. Am. M. Ass., January 4, 1930, xciv, 23.—(G. L. L.)*

Fusospirochaetal Disease of Lungs.—The various microbes present in the throat in cases of Vincent's angina are also constantly found in certain serious pulmonary lesions. Bacteriological studies are recorded in which some eight organisms were obtained in pure culture from the throats of patients with angina, and in which experiments were performed to determine the pathogenicity of these organisms. Material from 5 of 6 cases

of angina was successfully inoculated into guinea pigs, the material removed from the throat being transferred at once into a sterile petri dish containing 5 to 10 cc. of sterile horse serum, in which it was chopped up before being immediately injected into the groin of two guinea pigs. In fourteen days large foul-smelling abscesses were present, and these were comparable in all respects to those produced by the injection of sputum from cases of lung abscess and bronchiectasis. The infection was transmitted from guinea pig to guinea pig for 15 passages. Fresh pus from these abscesses was injected slowly into the trachea of 10 anaesthetized rabbits. Two died of pneumonia, three developed gangrene of the entire lung, four had lesions limited to one lobe of the lung and developed cavities, and of these two had a complicat ing empyema. The tenth rabbit survived, was given a second inoculation of pus, and then two doses of sulpharsphenamine. Three months later lipiodol injections and necropsy showed several dilated bronchi which resembled bronchiectasis. Dark-field examination of the material from a throat revealed four types of spirochaetes, a large and a small fusiform bacillus, a vibrio, and cocci. By aerobic and anaerobic cultural methods there were isolated a vibrio, an anaerobic haemolytic streptococcus, a large fusiform bacillus, a small fusiform bacillus, and *Treponema microdentium*. A mixture of the three latter organisms failed to produce any infection on injection into mice or guinea pigs. Other combinations gave inflammatory reactions of varying severity. Only when *Treponema microdentium*, the streptococcus, the small fusiform bacillus and the vibrio were injected together was a large, foul-smelling abscess produced. This was transmitted through a series of four guinea pigs, and when inoculated into the trachea of four rabbits produced lesions similar to those noted above. It is concluded from these experiments that fusospirochaetal disease of the lungs can be caused by the organisms commonly associated with Vincent's angina and that the pulmonary lesions are probably due to a symbiosis of a spirochaete, a fusiform bacillus, a vibrio and a coccus. It is quite possible that any of the spirochaetes of the mouth and a number of the cocci may replace the ones actually used in this experimental synthesis.—*Fusospirochetal Disease of the Lungs Produced with Cultures from Vincent's Angina, David T. Smith, J. Infect. Dis., April, 1930, xlvi, 303.*—(*R. D.*)

Nonputrid Pulmonary Necrosis.—The clinical history and roentgenological observations of 5 cases of nonputrid pulmonary necrosis are described. The clinical course and autopsy observations in 3 others are described. Nonputrid, pulmonary necrosis produces no symptoms. It is either discovered as a roentgenological observation or disclosed at autopsy. It is probably not an uncommon occurrence if looked for. Nonputrid pulmonary necrosis is differentiated from abscess of the lung complicating pneumonia by the absence of increased cough, foul sputum, and clubbing of the fingers, and by the fact that the cavity heals spontaneously in a relatively short time.—*The Clinical Aspect of Aputrid Pulmonary Necrosis, L. Kessel, Arch. Int. Med., March, 1930, xlv, 401.*—(*W. I. W.*)

Echinococcus Cysts of Chest-Wall.—This article is largely a review of the literature on echinococcus cysts of the chest-wall. It is an uncommon condition, occurring mostly in midadult life. The cysts are anatomically classified as (1) costal and sternal, (2) vertebrocostal, and (3) muscular, which may be primary or secondary. The primary costal cysts lodge usually at the dorsal extremity of the rib. The multilocular form appears as a result of the proliferation of the parasite or of a rupture of the original unilocular cyst. The vertebrocostal form arises by direct continuity from the ribs. The parasite gradually invades the structures around the spinal column which is soon eroded. This lesion is of diagnostic significance, as often it may be mistaken for Pott's disease of the dorsal vertebrae. As to symptoms, two stages can be distinguished; (1) the period of latency with no physical signs or symptoms and (2) an external stage when the tumor first appears, a phase of active proliferation of the parasite, which assumes within several months the size of a huge painless and multiloculated tumor. In both the costal and vertebral forms the X-ray readily discloses any abnormality. This is a valuable aid in establishing a diagnosis. Differential diagnosis rests mainly between echinococcus cyst, thoracic cold abscess, dorsal Pott's disease, and benign and malignant tumors. The first two are the commonest and most important. X-ray and diagnostic puncture for search for the characteristic hooklets will differentiate between a cyst and a cold abscess. In certain cases a diagnostic pneumothorax may be attempted to outline the boundaries of any intrathoracic tumor. It is especially emphasized that the diagnosis of a hydatid cyst should always be kept in mind in the presence of a noninflammatory cystic tumor. Treatment is purely surgical, involving the complete enucleation of all the vesicles. The prognosis in the vertebrocostal form, with symp-

toms of spinal-cord compression, is unfavorable, and only palliative treatment can be rendered.—*L'echinococcose de la paroi thoracique, H. Costantini and R. M. Marill, Arch. Méd.-Chir. d. l'Appar. Respir., July, 1929, iv, 331.*—(*J. G.*)

Inflammation of Diaphragm.—Inflammation in the neighborhood of and implicating the diaphragm is fairly common. The diaphragm is innervated by the phrenic nerves, which are distributed on its abdominal surface and are its sole motor supply. The sensory fibres of the phrenics are distributed over the central part of the abdominal surface, and irritation of these fibres may cause "shoulder-tip" pain, since the phrenic nerve arises from the same segment of the spinal cord as the cutaneous nerves of the supraclavicular region. Sensory fibres of the lower intercostal nerves are distributed to the more peripheral portion of the diaphragm, and irritation of these fibres may induce pain in the lower costal region and anterior abdominal wall. Acute diaphragmatic pleurisy is characterized by an abrupt onset, severe pain referred to the lower costal region, upper abdomen and supraclavicular region; vomiting; small and rapid pulse; impeded and frequently accelerated respirations; hyperalgesic skin over the painful area; absence of abdominal rigidity; and friction rub. Subacute diaphragmatic pleurisy does not cause as severe pain as the acute type. Chronic diaphragmatic pleurisy, in which old adhesions exist between the visceral pleura and the diaphragm, may not give rise to any symptoms and may be diagnosed only by X-ray examination. Frequently recurring sharp pain in the lower costal region, with occasional pain in the shoulder, is a help in diagnosis. There may be some impairment of movement of the diaphragm with diminished air entry at the base of the lung. Adhesions may extend between the diaphragmatic surface of the pleura and the diaphragm, particularly from the diaphragmatic part of the interlobar fissures, causing "tenting" of the diaphragm; along the periphery of the diaphragm so that the diaphragm is hooked up; or from the mediastinal surface of the lung to the diaphragm or pericardium. The most common causes for subdiaphragmatic abscess are perforation of a peptic ulcer and the "tracking up" of an appendicular abscess. The symptoms are languor, loss of appetite, emaciation, constipation, dry furred tongue, rapid pulse, pain in lower costal region, and impeded breathing. Difficulty may be experienced in differentiating it from pleural effusion. X-ray studies and exploration are often necessary in order to make a positive diagnosis. The prognosis is grave and the treatment is surgical.—*Inflammation of the Diaphragm, A. Ramsbottom, Brit. M. J., July 5, 1930, no. 3626, 5.*—(*M. E. P.*)

Gastric Deformities following Changes in Diaphragm.—In 1926 it was noted that morphological changes occurred in the contour of the stomach after left-sided phrenicectomy. The changes were transverse biloculation, anteroposterior biloculation, and the formation of oesophageal folds. Spasm of the musculature does not account for these anatomical variations, as they are not influenced by atropine. The stomach, spleen and diaphragm are joined by three ligaments: (1) that between the greater curvature of the stomach and the hilum of the spleen, (2) the suspensory ligament of the spleen, which attaches the latter organ to the cupola of the diaphragm, and (3) the suspensory ligament of the stomach between the diaphragm and the posterior surface of the stomach. It is believed that these gastric deformities are due to an unequal traction by these three peritoneal folds. Several X-rays are reproduced and an autopsy report is included in the article. The opinion is expressed that these deformities occur frequently after left-sided phrenicectomy. They cause no clinical symptoms and do not aggravate any preëxisting intestinal symptoms in tuberculosis patients. The physiological function of the stomach remains normal.—*Étude des déformations gastriques survenant à la suite des anomalies dans la forme et la situation du diaphragme, G. Dersheid and Toussaint, Arch. Méd.-Chir. d. l'Appar. Respir., September, 1929, iv, 389.*—(*J. G.*)

Dissecting Haematoma of Phrenic Nerve.—*Case Report:* A female child, aged sixteen months, was admitted to the hospital with bronchopneumonia. The first roentgenoscopic observation showed some restriction in the motion of both diaphragms. The roentgenograms in the anteroposterior view revealed a diffuse mottling of both lungs with a dense shadow at the right base, which in the lateral view gave the characteristic appearance of an interlobar collection between the right middle and lower lobes. A second roentgen examination was made five days later. There was a typical paradoxical movement of the right diaphragm, indicating a paralysis of the right phrenic nerve. The treatment consisted largely of fluids and transfusions. The day following the last roentgen examination of the chest the patient's condition became markedly worse and she died. At autopsy a dissect-

ing haematoma about the right phrenic nerve and an anomalous enlargement of the right lobe of the liver were found. The condition found at autopsy made clear the roentgen observations. The possibility of a paralysis of the diaphragm as a complication in the use of the jugular veins for the purpose of transfusions affords knowledge of distinct clinical value.—*Anomalous Enlargement of the Liver and a Dissecting Hematoma of the Phrenic Nerve, K. Kornblum and G. W. Stephenson, Am. J. Roentgenol & Rad. Ther., July, 1930, xxiv, 38.*—(*W. I. W.*)

Bronchomycosis.—Two cases are reported of extensive chronic interstitial fibrosis of the lungs in which tubercle bacilli were consistently absent. A form of yeast was obtained in both cases, which, in its morphology and cultural characteristics, resembled *Monilia albicans* as given in Castellani's table, with one exception: namely, that gelatine was not liquified. This organism when injected into white rats produced a pathological condition similar to that produced by the tubercle bacillus, but these lesions did not show any tendency to spread through the internal viscera, and simply remained either as local processes, or as scattered nodules on the serous surfaces.—*Bronchomycosis: Report of Two Cases, W. R. Stokes, E. F. Kiser and W. H. Smith, J. Am. M. Ass., July 5, 1930, xcv, 14.*—(*G. L. L.*)

Primary Cancer of Lung.—A report is made of 6 cases of primary pulmonary tumor, with necropsy. Primary cancer of the lung is now responsible for from 5 to 10 per cent of deaths from cancer. Carcinoma of the lung is more frequent than sarcoma. Cancer is more frequent in men than in women, and the incidence is highest in the sixth decade. It may be associated with lung abscess or tuberculosis. The cardinal symptoms are pain, which is more or less persistent, dyspnoea, cough, sputum and fever. Pleural effusion is frequently sanguineous, and occasionally purulent. According to the clinical classification of the author, there may be (1) slight or mild symptoms from a small solitary nodule; (2) severe symptoms from extensive pulmonary involvement; (3) symptoms due to invasion of, or pressure on, the mediastinum from extensive pulmonary involvement; and (4) symptoms from extensive metastatic areas from an asymptomatic tumor. A careful history, physical examination and roentgenogram will usually establish the diagnosis. Early surgical resection offers the only hope of cure. Roentgen therapy has afforded temporary palliation in some instances.—*Primary Lung Tumor: Report of Six Cases with Necropsies, L. M. Bonner, J. Am. M. Ass., April 5, 1930, xciv, 1044.*—(*G. L. L.*)

Primary Cancer of Lung.—The postmortem incidence of primary carcinoma of the lung is steadily increasing, and this increase is real and absolute. Combined statistics show that primary carcinoma of the lung at autopsy from 1910 to 1919 comprised 0.44 per cent of autopsies, and 4.39 per cent of all cancers. Since 1920, primary carcinoma of the lung at autopsy comprised 0.89 per cent of autopsies and 6.98 per cent of all cancers. Primary carcinoma of the lung is not as rare as was formerly believed. Because of its increased frequency, the clinician should give this affection serious consideration in differential diagnosis in patients of the carcinomatous age presenting puzzling pulmonary symptoms and signs. An early diagnosis will permit accurate prognosis, and in selected cases, perhaps, surgical therapy.—*The Incidence of Primary Carcinoma of the Lung, P. D. Rosahn, Am. J. Med. Sc., June, 1930, clxxix, 803.*—(*W. I. W.*)

Cancer and Pulmonary Tuberculosis.—Until recently pulmonary cancer was considered rare, yet from 1926 to April, 1928, Verstraeten personally observed four primary cases and there must have been many more secondary. Even necropsy will not always permit a positive diagnosis of the nature of the lesions. Microscopic examination is always necessary, especially as tuberculosis may be associated with the lesions. As to their actual frequency, Hunt says that primary pulmonary neoplasm is next most frequent after cancer of the stomach, breast, uterus and oesophagus. *General Symptoms:* Cachexia is very late in appearance, a terminal symptom. Absence of the slight elevations of temperature noted with tuberculosis is the rule, but not an absolute one. Cough is an early symptom, is dry, unproductive, and often spasmodic and painful. Sedatives and antispasmodics fail to affect it. There is often dyspnoea out of proportion to the objective symptoms. Haemoptyses occur in 50 per cent of cases but the blood congeals so as to look like gooseberry jelly. None of the patients observed by Verstraeten, either with primary or secondary cancer, have exhibited frank haemoptyses. *Objective Symptoms:* These may be divided into those revealed by physical signs and those revealed by X-ray. Of the former, a localized zone or area of dulness on percussion, suspended in the midst of a normal zone, is most important. When such a zone is found, X-ray examination will show an

opacity with well-defined limits. It is usually readily differentiated from opacity due to pleural exudate. Auscultation gives less useful information. Subclavicular adenopathy is rarely met with and can just as well be due to cancer of the stomach. An important, though usually late symptom, is the appearance of a haemorrhagic effusion containing cancerous cells. *Case Reports: 1:* A dressmaker, aged 57, was seen early in 1928 because of dry cough and dyspnoea. Examination of the chest showed at the right apex a clearly localized zone of dulness. There were also fine, inconstant, dry râles. Roentgenographs showed at the right apex a localized opacity, and at the left small disseminated pulmonary nodules. Tubercle bacilli were not found in the sputum. Cancer was diagnosed. Two applications of roentgenotherapy were given in April, 1928, following which the mass shrunk and some improvement was noted. Toward the end of 1928 she was seen again and now showed dulness and râles over the entire right lung. The sputum now showed definitely cancerous cells, together with many large flattened epithelial cells. In May, 1929, the patient developed an exudate. Roentgenographs showed metastatic nodules in the left lung and a haemorrhagic exudate developed there within a few days. Roentgenotherapy again caused amelioration but she died in June, 1929. The right lung at necropsy was found to be symphysized and to contain a vast cavity involving upper and middle lobes. The remainder of it was collapsed. The left lung showed multiple nodules, white, of viscid consistency, containing necrotic centres filled with pus and coagulated blood. No metastases were found in other organs. Histological examination showed the walls of the large cavity plastered with numerous cancerous cells, but also some tuberculous granulations and giant cells. *2:* This was a woman of 53, whose work had exposed her for several years to powdered lead. She appeared to have an attack of grippe in 1922, since when she had cough, expectoration and dyspnoea. On examination of the chest there was a zone of dulness limited to the middle third of the right lung, and also, on the left, a similar but smaller area. Roentgenographs showed in both these areas opaque masses with clearly defined contours. The sputum was black, anthracotic, consistently sanguinolent, and mucopurulent; it also showed tubercle bacilli after repeated examinations. The Wassermann test was positive. Although this case at first looked like pulmonary cancer it proved to be one of pulmonary tuberculosis complicated by anthracosis and syphilis, showing the need of careful differential diagnosis.—*Cancer et tuberculose pulmonaire, A. Verstraeten, Rev. Belge d. l. Tuberc., September-October, 1929, xx, 229.*—(*A. P.*)

Tuberculosis and Cancer.—May the tubercle bacillus determine neoplastic lesions? Many workers have seen a similarity in the cellular reactions and some were convinced of a relationship. To-day not much consideration is given this idea but the discovery of bacillary metamorphosis and change in character has had its effect. There may be tuberculous processes without giant cells and giant cells without tuberculosis. Certain facts of gross pathology are recalled. Tavernier exhibited a young man with tuberculous polyps of the intestine, simulating polyadenoma. Hartman has published cases of "pseudoneoplastic" renal tuberculosis. Mauclair and Vigneron cited a case of testicular tuberculosis of pseudoneoplastic type. In avian tuberculosis German and Raymond find quite a variety of types, with one resembling encephaloid carcinoma. Uncomplicated tuberculous lupus provokes modifications in the epithelial cells comparable to cancer, especially the sclerous type of lupus, and Busch describes a *lupus-carcinome.* In this case Ribbert directly accuses tuberculous toxins. Symbiosis of tuberculosis and carcinoma has been noted by Broca, Landouzy and Lebert. Of interest also are the facts attached to leukaemia. Maude Slye considers chronic leukaemia and pseudoleukaemia as following the lines of cancerous conditions and as neoplastic diseases. Myeloid transformation of the spleen, in this connection, has been noted in tuberculized guinea pigs. Aubertin has found a splenic anaemia, with myelocytes, and nucleated haematocytes, due to acute tuberculosis. That is, it has provoked a strong hyperplasia. Why may not the same hold true for connective-tissue cells? As an example of what a bacterial irritant may do, we have crown-gall, studied by Smith. The histological beginning of cancer and tuberculosis has similarities. Lymphoid metaplasia is common to both. Cancerous tissue, like tuberculous, shows an anarchic specialized reaction of the connective tissue. The question of the development of cancer on the cicatricial tissue of lupus is of some importance and not very infrequent. Claude and Ribbert both accuse the primary inflammatory process of being implicated in this. The fact is that cancer develops on fibrous tissue, which is often of tuberculous origin. In both conditions red blood cell sedimentation is accelerated. There is also a hyperglycaemia. Certain chemical bodies tend to induce cancer: for instance, aniline, benzidine,

indol. Are these not of similar derivation and one (indol) related to constitutional changes? Indol is a phenopyrrol and exists in oils of coal-tar derivation. Does the tubercle bacillus produce indol in certain media, like *B. proteus* or *B. coli*? Eighty-eight cases are cited in which there existed either cancer and tuberculosis in the same individual or in others of the family. One case is of particular interest. He had cancer of the pylorus with tubercles and cavities in the left upper lobe. His father died of cancer of the pylorus, both brothers died of cancer, and his son of tuberculosis. Does cancer develop on a tuberculous terrain? Can a modified tubercle bacillus, rich in erepsin, excite the cell to anarchic division, such as takes place in cancer? One must seek the solutions of these questions in variations of the physicochemical media. Recently Centenni and Modène and also F. Rezzeti have written on the same subject and it is necessary to refute their notion of an antagonism between tuberculosis and cancer. Of 100 cancerous families investigated by Burdet, 75 per cent had tuberculous antecedents. Riffel found that 86 per cent of subjects with cancer of the stomach had such antecedents and Roger Williams concludes that the descendants of the tuberculous are most apt to die of cancer. Experimental work with tumors in mice is cited, which however seems little to the point. It is concluded that it is necessary to undertake experimental investigations with attenuated tubercle bacilli grown on various kinds of media.—*La tuberculose et le cancer, J. Roux, Rev. d. l. Tuberc., August, 1929, x, 488.*—(*A. P.*)

Cerebrospinal Metastases from Cancer of Lung.—No satisfactory explanation has been given for the comparatively recent increase in the incidence of primary pulmonary carcinoma, which is now about 4 per cent of all carcinomata. The vigorous metastasizing power of lung carcinoma has been attributed to the lung's vascularity and abundant lymphatic channels, and to frequent invasion of blood-vessels by tumor. Necropsies at the National Hospital, London, during the past ten years show that of 29 tumors whose metastases produced signs of cerebrospinal involvement, nine were of pulmonary origin. Cerebrospinal involvement was found by Dosquet in 37 per cent of 105 cases, and by Fried in 47.4 per cent of 19 cases of pulmonary carcinoma. Possible success of radical surgery, or of radium therapy, for the primary lung lesion, necessitates closer investigation of these cases. Symptoms may point toward lung or cerebrospinal system, or both, or the case may die in coma without definite implication of either, though all the necropsies show involvement of both. In most cerebral cases symptoms are due to intracerebral deposits and only occasionally to secondary pressure from cranial (bone) metastases. In the spinal group only 7 or 8 cases of true intramedullary secondary deposits are on record, and in most of these symptoms were due to pressure from metastases in the vertebrae. Nine verified cases of primary pulmonary carcinoma with metastases causing signs of cerebrospinal involvement, treated in the National Hospital during the past three years, are described in detail. The necropsy findings may be summarized as follows: In all but one case the primary tumor was situated in or near the hilum of the lung. The tumors were firm and white, and in seven cases were found on the right side. Evidence of bronchial obstruction, that is, pulmonary collapse or bronchiectasis, was present in 6 cases. The tumor cells, markedly pleomorphic in nature, were arranged in solid trabeculae and showed a tendency to the formation of acini. Between the trabeculae were strands of fibrous tissue which carried blood-vessels. The cell nuclei were large and vesicular and many mitotic figures were seen. In 3 cases of this series, having symptoms from secondary deposits confined to bone and not invading nervous tissue, the "hollowed out and oyster shell" appearance on the inner aspect of the skull and vertebrae was conspicuous. Meningeal invasion probably accounts for the presence of radicular pain in some of the cases. The intracerebral tumors were strawberry-like in appearance, with a surrounding zone of haemorrhage, sharply demarcated from the brain tissue. Microscopically, the secondary deposits resembled the primary tumors. Pictures of the gross and microscopic pathology of the tumors are included.—*Cerebrospinal Metastases from Unsuspected Pulmonary Carcinoma, F. R. Ferguson and W. E. Rees, Lancet, April 5, 1930, ccxviii, 738.*—(*R. M. F.*)

Cancer of Lung after Trauma.—The difficulties in establishing the causal relation of trauma to tumors is pointed out. The literature reveals few instances in which a primary pulmonary carcinoma has seemed to result from a single trauma to the lung tissue; hence any apparently probable relationship between a single trauma and a subsequent tumor should be placed on record for future investigators of the problem. The present case report is of special value because the X-ray plates of the site of the injury made immediately after the injury was received showed that at that time there was no recognizable evidence of malignant

disease. *Report of Case:* A man, aged 50, in good general health, was knocked down by an automobile. An X-ray examination the next day revealed fractures of the left third, fourth and fifth ribs in the midaxillary line. The films showed no evidence of any neoplasm in the lung. There was also blood-spitting and a subcutaneous emphysema extending over the entire body, indicating traumatic injury of the lung. The fractures healed and the patient seemed to be in good health until eleven months later when he began to have pain in the left side of his chest, accompanied by cough and symptoms suggestive of tuberculosis. No tubercle bacilli were ever found in the sputum. X-ray films now revealed evidence of cancer in the left upper lobe with shadows in the hilar area suggesting metastases to the lymph nodes. Repeated bronchoscopic examinations were negative. The clinical course was steadily downward and death occurred one year after the development of symptoms, and just short of two years after injury to the left lung. The family history revealed that a paternal grandmother died of carcinoma of the breast and that a paternal uncle died of carcinoma of the stomach. The necropsy revealed the presence of a primary tumor in the upper lobe of the left lung, with metastases in the mediastinal and left supraclavicular lymph nodes, in the retroperitoneal periaortic lymph nodes as far down as the bifurcation, in the right suprarenal gland and both kidneys. The upper lobe of the left lung was found to be almost completely replaced by a dense white tumor mass. There was no growth in the trachea or either primary bronchus, nor in the large branches of the bronchi on either side. Gross examination indicated that the tumor arose in the periphery of the left upper lobe, rather than in one of the larger bronchi, where primary carcinoma of the lung usually has its origin. Histologically the tumor resembled sarcoma, for in no places did the cells exhibit a characteristic epithelial structure. The structure was that of the type of lung tumors described as arising from the alveoli. While it is certainly not possible to determine by the histological character of a pulmonary carcinoma whether it arose in the bronchi or in the alveoli, the absence of gland-like structures or of mucin and the tendency to the formation of spindle cells resembling a sarcoma are points which suggest the peripheral origin of the tumor. Neither is it possible to say that there was not already a carcinoma, too small to be detected in the X-ray film, growing in the part of the lung that was traumatized at the time of the injury. But to support such an explanation of the particular case requires a stretching of "the long arm of coincidence" to the vanishing point. Absolute anatomical proof can never be furnished that a given tumor arising at the site of a trauma has resulted from the tissue damage or subsequent reparative proliferation of previously normal tissues. It is equally impossible to prove that any tumor arising at the site of and subsequent to an injury is not the result of tissue damage or subsequent reparative proliferation, if there has been no demonstrated tumor at this site at or before the time of the traumatism. The case herewith reported seems to present as nearly completely satisfactory evidence as one can hope to secure of the development of a primary carcinoma of the lung as a direct result of a single traumatism to the lung tissue.—*Primary Carcinoma of the Lung following Trauma, H. G. Wells and P. R. Cannon, Arch. Path., April, 1930, ix, 869.* (*J. S. W.*)

Mediastinal Lipoma.—*Case Report:* A white man, aged forty-four, had moderate dyspnoea on exertion for eleven years, and he died of lobar pneumonia. Postmortem examination revealed a lipoma. It weighed 8¼ pounds and filled the anterior mediastinum, the pericardium, and the lower two-thirds of the right thoracic cavity.—*Lipoma of the Mediastinum, W. M. Yates and E. S. Lyddane, Am. J. Med. Sc., July, 1930, clxxx, 79.*—(*W. I. W.*)

Sarcoma of Rib.—Sarcoma of the rib is to be differentiated from tuberculosis, which can be done only with great difficulty, because the tumor may be only a destructive process resembling tuberculosis. In young individuals the history of injury associated with pain should suggest a tumor. Palliative roentgen therapy should be used in all cases as a means of relieving pain and prolonging life —*Sarcoma of the Rib, R. J. Reeves and H. H. Kasabach, Am. J. Roentgenol & Rad. Ther., September, 1930, xxiv, 262.*—(*W. I. W.*)

Lymphoblastoma, Hodgkin's Disease and Tuberculosis.—A man, 61 years of age, had enlargement of all the superficial lymph nodes and a leucocyte count of 11,600 with 44 per cent lymphocytes. A biopsy specimen was diagnosed lymphoblastoma of the type consistent with aleukaemic leukaemia. A year and a half later the patient showed a general lymph-node enlargement, and distention of the abdomen due to the presence of fluid was marked. The white blood cells were 60,000 per cmm., with 90 per cent lymphocytes. A diagnosis of

lymphatic leukaemia was made. An acute infection of an inguinal lymph node developed and demanded drainage. After the temperature returned to normal the white blood count was only 10,000 and the differential was repeatedly normal. More than a year elapsed after this before the patient died and during this time the blood count remained essentially normal. At autopsy the peritoneum contained 4,000 cc. of milk-like fluid which on animal inoculation was found to contain tubercle bacilli of the human type. The lungs on section showed old and recent tuberculous lesions. Both the liver and spleen showed tubercles, discrete areas composed entirely of cells resembling those of the Hodgkin's type of tumor cell, and rather discrete foci of mature lymphocytes. The jejunum contained an ulcer on whose surface were miliary tubercles. Miliary tubercles and small foci of mature lymphocytes occurred in the bone-marrow of the vertebrae. The lymph nodes showed a wide variation. In some there were as many as three distinct pathological lesions. The diagnoses were lymphoblastoma, Hodgkin's disease and tuberculosis. The possible interrelation of the three conditions is discussed. The frequent association of Hodgkin's disease with tuberculosis has been the subject of many papers. One of the most recent of these, by L'Esperance, suggests that the avian tubercle bacilli may be a factor in producing some of the lesions interpreted as Hodgkin's disease. In the case here reported the tuberculous lesions in the lungs were of long standing, and it is possible that the tubercle bacillus may have borne some relation to the initiation of the original tumor of the lymphatic apparatus, and even more probable that it modified the subsequent cytological and histological course. However, these are simply hypotheses which still remain to be proved.—*A Case of Lymphoblastoma, Hodgkin's Disease, and Tuberculosis, H. E. MacMahon and F. Parker, Jr., Am. J. Path., May, 1930, vi, 367.*—(*R. D.*)

Intestinal Lymphogranulomatosis.—Isolated lymphogranulomatosis of the gastrointestinal tract is relatively rare. Since Schlagenhaufer published the first case, about 30 cases of this type of disease have been recorded in the literature. According to Fränkel, the intestinal lesions were classified into three groups: (1) tumor-like masses, which protrude into the lumen; (2) flat, circumscribed infiltrations of the wall; and (3) diffuse infiltrations, which frequently may involve large parts. According to the various pathologicoanatomical types, the clinical course shows marked variations. In some cases the infectious process as such, in others the localized lesions are chiefly responsble for the symptomatology. In the tumor-like group fever is rare, while in the other groups, especially in the third one, recurrent attacks of fever or continuous fever is a common occurrence. There is no haematological picture characteristic of the disease. In the advanced stages an absolute leucopoenia with a relative lymphopoenia is frequently observed, while in the more incipient cases an absolute leucocytosis is sometimes observed. Increase in monocytes and eosinophilic cells is reported. The disease always produces a secondary anaemia. Skin lesions are less frequent in intestinal lymphogranulomatosis than in the generalized type. Occasionally, the following processes have been found: necroses, ulcerations, psoriasis, pityriasis, dermatitis and brownish discolorations. Indicanuria and albuminuria are observed occasionally and the diazo reaction may become positive. Patients with a chronic clinical picture have usually isolated, tumor-like lesions, and the symptomatology is that of a slow-growing intestinal tumor. If the mass is localized in the stomach or duodenum, vomiting and progressive loss of weight occur. There may be an achlorhydria, gastric retention, and presence of lactic acid in the gastric contents. The X-ray picture shows the same characteristics as those of a gastric carcinoma. In 2 cases pyloric stenosis was found. Tumor masses in the upper intestinal tract make similar symptoms and are apt to lead to stenosis. The lesions are relatively frequently found near the ileocaecal valve. In such cases, the differential diagnosis between lymphogranulomatosis and recurrent chronic appendicitis may be impossible. Clinically acute diseases are caused by diffuse infiltrative forms. The symptoms may resemble those of typhoid fever. Since there may be a marked leucopoenia, the differential diagnosis is difficult. Haemorrhage from ulcerated lesions, and perforation with subsequent intraperitoneal abscesses or generalized peritonitis are reported. Although temporary improvements have been noticed after the removal of localized lesions, it must be assumed that the final prognosis is not any better than in other cases of lymphogranulomatosis.—*Zur Klinik der intestinalen Lymphogranulomatose, R. Klima, Wien. klin. Wchnschr., December 19, 1929, xlii, 1626.*—(*M. P.*)

Lymphadenoma Not Tuberculous.—A series of patients suffering from lymphadenoma was tested with intracutaneous tuber-

culin in an effort to secure evidence for or against the theory that the disease represents an atypical form of tuberculosis. At the same time other patients were tested as a check on the method. Old Tuberculin was diluted 1:1000, and used while the dilution was fresh. Intracutaneous injection of 0.1 cc. into the skin of the forearm was carried out, and a similar amount of control solution was injected at a distance of 2 inches. The test was read at the end of 48 hours. The positive reactions showed a central rather pale area of swelling, with a surrounding area of erythema. Occasionally simple erythema occurred, and the cases were found to belong to the doubtful or nontuberculous groups. More seldom was there simple swelling, and here the association was with proved tuberculosis. It is concluded that attention should be paid to the area of swelling only. An area less than 1 cm. in diameter was read as "weak positive"; between 1 and 2 cm. as "positive"; any reaction in excess of this as "strongly positive". *Results: Group 1: Cases of Proved Tuberculosis:* 70 of 76 were positive. *Group 2: Cases Almost Certainly Tuberculous:* 50 of 58 were positive. *Group 3: Cases with No Evidence of Tuberculosis:* 59 of 189 were positive. *Group 4: Cases Suffering from Lymphadenoma:* 7 of 42 were positive. In three of the positive and one of the negative cases there was evidence of the coexistence of tuberculosis. If any significance is to be attached to the test at all, the tubercle bacillus apparently plays no part in the causation of lymphadenoma.—*The Intradermal Tuberculin Test, J. Maxwell, Lancet, August 9, 1930, ccxix, 289.* (*R. D.*)

Recoveries from Leprosy.—A report recently issued by the Public Health Service gives an interesting summary of the value of medical treatment for leprosy at the National Leprosarium which is conducted by the Public Health Service at Carville, La. More than 300 lepers, men, women and children, are under treatment there. During the past ten years, 65 lepers have been discharged from this hospital as apparently recovered from leprosy and no longer a menace to the public health. The average period of hospital care varied from 5 to 9 years. The shortest period of treatment was 1½ years and the longest was 17 years. Fifty-five of these patients received crude chaulmoogra oil by mouth, and 16 of this group received no other medicine. Twelve received benzocaine-chaulmoogra oil by intramuscular injection, and 4 of these received no other medical treatment. Twenty-one received the ethyl esters of chaulmoogra oil by intramuscular injection, and 8 of these received no other medicine. The basic treatment of leprosy is similar to that for tuberculosis, and all lepers at the National Leprosarium, no matter what medicines are given, follow a sanatorium regimen of food, fresh air and rest,—almost identical with that prevailing in a tuberculosis hospital.—*Health News, U. S. Public Health Service, leaflet, November 8, 1930.*

Intracutaneous Tests on Lepers.—Intracutaneous tests with extract from leprosy lesions were made in cases of leprosy and in nonlepers. The object of this study was to determine whether "latent leprosy" may be a factor in the percentage of tuberculin positives in South African natives. The data collected revealed a considerably lower allergic response of the nonleprous groups to the leprosy extract as compared with the leprous groups. This evidence is against the probability of the latent leprosy factor playing any considerable part in the high reactivity of healthy natives to tuberculin.—*Intradermal Tests with Extract of Leprous Skin in Cases of Leprosy and in Non-Lepers, S. L. Cummins and J. J. D. Le Roux, Tubercle, April, 1930, xi, 299.*—(*W. I. W.*)

Erythema Nodosum.—There is an undeniable association between erythema nodosum and tuberculosis. The evidence in support of this opinion is strong on the clinical side. Tuberculin tests and skiagraphy, in the hands of experts, afford confirmatory evidence of high value. The pathological and bacteriological evidence is weak.—*Erythema Nodosum and Tuberculosis, J. O. Symes, Tubercle, January, 1930, xi, 154.*—(*W. I. W.*)

Tuberculosis and Schizophrenia.—Some French and Spanish writers maintain the idea that schizophrenia is a tuberculous disease. Luxemburger, however, believes that a positive correlation exists between the hereditary tendency to the two diseases. This opinion is based on the fact that he was able to show that the nonpsychotic blood relatives of schizophrenic patients have a considerably higher tuberculosis mortality than the general population. The authors show that the leptosomous constitution occurs about as frequently in schizophrenic as in tuberculosis patients. It is not justified to conclude from such observations that the two diseases have a tendency to coexist. It is more likely correct to assume that the leptosomous habitus has an affinity both to tuberculosis and to schizophrenia. This would explain the higher tuberculosis mortality among relatives of schizophrenics,

since these relatives as well as the patients are more frequently of leptosomous build.—*Tuberkulose und Schizophrenie, K. Westphal and M. H. Welti, Klin. Wchnschr., May 31, 1930, ix, 1025.*—(*M. P.*)

Tuberculosis and Diabetes.—The complication of pulmonary tuberculosis is a serious one for the patient with diabetes. At the present time there are probably in the neighborhood of sixteen thousand diabetics in this country afflicted with active pulmonary tuberculosis, and of these more than half are likely to die within the first twelve months after the correct diagnosis is established. Pulmonary tuberculosis is particularly prone to develop in the elderly diabetic, of poor circumstances, leading a sedentary life, who has been at one time obese and through inadequate treatment for diabetes has lost weight rapidly. The tuberculosis may begin deep in the hilar region of the lung, and at first may cause no signs or symptoms. Before long, however, the tuberculous diabetic will develop the usual signs of pulmonary tuberculosis with positive sputum. The pulmonary lesion in its beginning may be unrecognizable except by the roentgen ray, but later can be detected easily by ordinary physical diagnosis. Tuberculosis may develop in a diabetic patient while the patient is under treatment. It is very important for any doctor or clinic, treating diabetic patients, to remember this, and to be on the lookout for a developing pulmonary tuberculosis. A patient under treatment for diabetes who does not hold his weight or who does not appear to be getting along satisfactorily should be suspected of having tuberculosis. All diabetic patients should be watched carefully and should have repeated examinations from time to time, in order to forestall further complications. The treatment of pulmonary tuberculosis according to the experience of the Peter Bent Brigham Hospital cases is unsatisfactory. At best, it is time-consuming and expensive, for it requires unlimited rest, fresh air, and, in addition, adequate diabetic treatment for a long period of time. There may be apparent temporary improvement under any appropriate therapy, but the final prognosis is poor. The cases which lived the longest were those which had a quiescent pulmonary process antedating the diabetes. Later, this became active, but was more amenable to treatment than the fresh type of tuberculosis developing as a later complication of diabetes in an unresistant individual.—*The Problem of Pulmonary Tuberculosis in Patients with Diabetes, R. Fitz, Am. J. Med. Sc., August, 1930, clxxx, 701.*—(*W. I. W.*)

Outcome of Pulmonary Tuberculosis.—The author reports on 636 open cases of tuberculosis of the first stage (Turban I) that were observed during the years 1912, 1913 and 1914, and in 340 suitable follow-up information was obtained. The patients were divided into children up to 14 years, from 14 to 19 years, and then from then on in decades. The fate of the patients after 5, 10 and 15 years is recorded. The course of the disease is most favorable in children. The outcome of the cases as a whole was fairly favorable.—*Über die Ausgänge der Lungentuberkulose. (Turban I), E. R. Kritschewskaja, Beitr. z. Klin. d. Tuberk., 1929, lxxii, 79.*—(*H. J. C.*)

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